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RESPIRATORY CARE

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47TH YEAR—ESTABLISHED 1956

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EDITORIALS

Evidence-Based Practice Guidelines for Ventilator Weaning

ORIGINAL CONTRIBUTIONS

Albuterol Administration Practices in Intubated Patients in the Newborn ICU

Work of Breathing in Spontaneously Breathing Animals During Partial Liquid Ventilation

Effects of Motion Artifact and Low Perfusion on the Performance of a New Generation of Pulse Oximeters

Modification of a Critical Care Ventilator for Use During Magnetic Resonance Imaging

SPECIAL ARTICLES

ACCP/AARC/SCCM Evidence-Based Guidelines for Weaning and Discontinuing Ventilatory Support

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE

DuoNeb is indicated for the treatment of bronchospasm associated with COPD in patients requiring more than one bronchodilator

CONTRAINDICATIONS

DuoNeb is contraindicated in patients with a history of hypersensitivity to any of its components, or to atropine and its derivatives

WARNINGS

Paradoxical Bronchospasm: In the clinical study of DuoNeb, paradoxical bronchospasm was not observed However, paradoxical bronchospasm has been observed with both inhaled ipratropium bromide and albuterol products and can be life-threatening. If this occurs, DuoNeb should be discontinued immediately and alternative therapy instituted

Do Not Exceed Recommended Dose: Fatalities have been reported in association with excessive use of inhaled products containing sympathomimetic amines and with the home use of nebulizers

Cardiovascular Effect: DuoNeb, like other beta adrenergic agonists, can produce a clinically significant cardiovascular effect in some patients as measured by pulse rate, blood pressure, and/or symptoms Although such effects are uncommon for DuoNeb at recommended doses, if they occur, the drug may need to be discontinued In addition, beta agonists have been reported to produce ECG changes, such as flattening of the T-wave, prolongation of the QTc interval, and ST segment depression. The clinical significance of these findings is unknown. Therefore, DuoNeb, like other sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension

Immediate Hypersensitivity Reactions: Immediate hypersensitivity reactions to albuterol and/or ipratropium bromide may occur after the administration of DuoNeb as demonstrated by rare cases of urticaria, angioedema, rash, pruritus, oropharyngeal edema, bronchospasm, and anaphylaxis.

PRECAUTIONS

General

1 Effects Seen with Sympathomimetic Drugs: As with all products containing sympathomimetic amines, DuoNeb should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension; in patients with convulsive disorders, hyperthyroidism, or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic amines Large doses of intravenous albuterol have been reported to aggravate pre-existing diabetes mellitus and ketoacidosis Additionally, β-agonists may cause a decrease in serum potassium in some patients, possibly through intracellular shunting The decrease is usually transient, not requiring supplementation.

2 Effects Seen with Anticholinergic Drugs: Due to the presence of ipratropium bromide in DuoNeb, it should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy, or bladder-neck obstruction

3. Use in Hepatic or Renal Diseases: DuoNeb has not been studied in patients with hepatic or renal insufficiency It should be used with caution in these patient populations

Information for Patients

The action of DuoNeb should last up to 5 hours DuoNeb should not be used more frequently than recommended. Patients should be instructed not to increase the dose or frequency of DuoNeb without consulting their healthcare provider If symptoms worsen, patients should be instructed to seek medical consultation

Patients must avoid exposing their eyes to this product as temporary papillary dilation, blurred vision, eye pain, or precipitation or worsening of narrow-angle glaucoma may occur, and therefore proper nebulizer technique should be assured, particularly if a mask is used

If a patient becomes pregnant or begins nursing while on DuoNeb, they should contact their healthcare provider about use of DuoNeb

See the illustrated Patient's Instruction for Use in the product package insert

Drug Interactions

Anticholinergic agents: Although ipratropium bromide is minimally absorbed into the systemic circulation, there is some potential for an additive interaction with concomitantly used anticholinergic medications. Caution is, therefore, advised in the co-administration of DuoNeb with other drugs having anticholinergic properties.

β-adrenergic agents. Caution is advised in the co-administration of DuoNeb and other sympathomimetic agents due to the increased risk of adverse cardiovascular effects.

β-receptor blocking agents. These agents and albuterol sulfate inhibit the effect of each other β-receptor blocking agents should be used with caution in patients with hyperreactive airways, and if used, relatively selective β_1 selective agents are recommended.

Diuretics. The electrocardiogram (ECG) changes and/or hypokalemia that may result from the administration of non-potassium sparing diuretics (such as loop or thiazide diuretics) can be acutely worsened by β-agonists, especially when the recommended dose of the β-agonist is exceeded. Although the clinical significance of these effects is not known, caution is advised in the co-administration of β-agonist-containing drugs, such as DuoNeb, with non-potassium sparing diuretics.

Monoamine oxidase inhibitors or tricyclic antidepressants. DuoNeb should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants, or within 2 weeks of discontinuation of such agents because the action of albuterol sulfate on the cardiovascular system may be potentiated

Carcinogenesis, Mutagenesis, Impairment of Fertility

Albuterol Sulfate: In a 2-year study in Sprague-Dawley rats, albuterol sulfate caused a significant dose-related increase in the incidence of benign leiomyomas of the mesovarium at and above dietary doses of 2 mg/kg (approximately equal to the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). In another study, this effect was blocked by the coadministration of propranolol, a non-selective beta-adrenergic antagonist.

In an 18-month study in CD-1 mice, albuterol sulfate showed no evidence of tumorigenicity at dietary doses up to 500 mg/kg (approximately 140 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). In a 22-month study in Golden hamsters, albuterol sulfate showed no evidence of tumorigenicity at dietary doses up to 50 mg/kg (approximately 20 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis)

Albuterol sulfate was not mutagenic in the Ames test or a mutation test in yeast Albuterol sulfate was not clastogenic in a human peripheral lymphocyte assay or in an AH1 strain mouse micronucleous assay.

Reproduction studies in rats demonstrated no evidence of impaired fertility at oral doses of albuterol sulfate up to 50 mg/kg (approximately 25 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis)

Ipratropium bromide. In 2-year studies in Sprague-Dawley rats and CD-1 mice, ipratropium bromide showed no evidence of tumorigenicity at oral doses up to 6 mg/kg (approximately 15 times and 8 times the maximum recommended daily inhalation dose for adults in rats and mice respectively, on a mg/m^2 basis).

Ipratropium bromide was not mutagenic in the Ames test and mouse dominant lethal test. Ipratropium bromide was not clastogenic in a mouse micronucleous assay

A reproduction study in rats demonstrated decreased conception and increased resorptions when ipratropium bromide was administered orally at a dose of 90 mg/kg (approximately 240 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis) These effects were not seen with a dose of 50 mg/kg (approximately 140 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis)

Pregnancy

TERATOGENIC EFFECTS: Pregnancy Category C

Albuterol sulfate. *Pregnancy Category C.* Albuterol sulfate has been shown to be teratogenic in mice A study in CD-1 mice given albuterol sulfate subcutaneously showed cleft palate formation in 5 of 111 (4.5%) fetuses at 0.25 mg/kg (less than the maximum recommended daily inhalation dose for adults on a mg/m^2 basis) and in 10 of 108 (9.3%) fetuses at 2.5 mg/kg (approximately equal to the maximum recommended daily inhalation dose for adults on a mg/m^2 basis) The drug did not induce cleft palate formation when administered subcutaneously at a dose of 0.025 mg/kg (less than the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). Cleft palate formation also occurred in 22 of 72 (30.5%) fetuses from females treated subcutaneously with 2.5 mg/kg isoproterenol (positive control).

A reproduction study in Stride rabbits revealed cranioschisis in 7 of 19 (37%) fetuses when albuterol was administered orally at a dose of 50 mg/kg (approximately 55 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis)

A study in which pregnant rats were dosed with radiolabeled albuterol sulfate demonstrated that drug-related material is transferred from the maternal circulation to the fetus

During worldwide marketing experience, various congenital anomalies, including cleft palate and limb defects, have been reported in the offspring of patients being treated with albuterol. Some of the mothers were taking multiple medications during their pregnancies Because no consistent pattern of defects can be discerned, a relationship between albuterol use and congenital anomalies has not been established

Ipratropium bromide. *Pregnancy Category B* Reproduction studies in CD-1 mice, Sprague-Dawley rats and New Zealand rabbits demonstrated no evidence of teratogenicity at oral doses up to 10, 100, and 125 mg/kg, respectively (approximately 15, 270, and 680 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). Reproduction studies in rats and rabbits demonstrated no evidence of teratogenicity at inhalation doses up to 1.5 and 1.8 mg/kg, respectively (approximately 4 and 10 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). There are no adequate and well-controlled studies of the use of DuoNeb, albuterol sulfate, or ipratropium bromide in pregnant women. DuoNeb should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Labor and Delivery

Oral albuterol sulfate has been shown to delay preterm labor in some reports. Because of the potential of albuterol to interfere with uterine contractility, use of DuoNeb during labor should be restricted to those patients in whom the benefits clearly outweigh the risks

Nursing Mothers

It is not known whether the components of DuoNeb are excreted in human milk. Although lipid-insoluble quaternary bases pass into breast milk, it is unlikely that ipratropium bromide would reach the infant to an important extent, especially when taken as a nebulized solution Because of the potential for tumorigenicity shown for albuterol sulfate in some animals, a decision should be made whether to discontinue nursing or discontinue DuoNeb, taking into account the importance of the drug to the mother

Pediatric Use

The safety and effectiveness of DuoNeb in patients below 18 years of age have not been established

Geriatric Use

Of the total number of subjects in clinical studies of DuoNeb, 62 percent were 65 and over, while 19 percent were 75 and over No overall differences in safety or effectiveness were observed between these subjects and younger subjects, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out.

ADVERSE REACTIONS

Adverse reaction information concerning DuoNeb was derived from the 12-week controlled clinical trial

ADVERSE EVENTS OCCURRING IN ≥ 1% OF ≥ 1 TREATMENT GROUP(S) AND WHERE THE COMBINATION TREATMENT SHOWED THE HIGHEST PERCENTAGE

Body System COSTART Term	Albuterol n (%)	Ipratropium n (%)	DuoNeb n (%)
NUMBER OF PATIENTS	761	754	765
N (%) Patients with AE	327 (43.0)	329 (43.6)	367 (48.0)
BODY AS A WHOLE			
Pain	8 (1.1)	4 (0.5)	10 (1.3)
Pain chest	11 (1.4)	14 (1.9)	20 (2.6)
DIGESTIVE			
Diarrhea	5 (0.7)	9 (1.2)	14 (1.8)
Dyspepsia	7 (0.9)	8 (1.1)	10 (1.3)
Nausea	7 (0.9)	6 (0.8)	11 (1.4)
MUSCULO-SKELETAL			
Cramps leg	8 (1.1)	6 (0.8)	11 (1.4)
RESPIRATORY			
Bronchitis	11 (1.4)	13 (1.7)	13 (1.7)
Lung Disease	36 (4.7)	34 (4.5)	49 (6.4)
Pharyngitis	27 (3.5)	27 (3.6)	34 (4.4)
Pneumonia	7 (0.9)	8 (1.1)	10 (1.3)
UROGENITAL			
Infection urinary tract	3 (0.4)	9 (1.2)	12 (1.6)

Additional adverse reactions reported in more than 1% of patients treated with DuoNeb included constipation and voice alterations

In the clinical trial, there was a 0.3% incidence of possible allergic-type reactions, including skin rash, pruritus, and urticaria.

Additional information derived from the published literature on the use of albuterol sulfate and ipratropium bromide singly or in combination includes precipitation or worsening of narrow-angle glaucoma, acute eye pain, blurred vision, paradoxical bronchospasm, wheezing, exacerbation of COPD symptoms, drowsiness, aching, flushing, upper respiratory tract infection, palpitations, taste perversion, elevated heart rate, sinusitis, back pain and sore throat

DOSAGE AND ADMINISTRATION

The recommended dose of DuoNeb is one 3 mL vial administered 4 times per day via nebulization with up to 2 additional 3 mL doses allowed per day, if needed Safety and efficacy of additional doses or increased frequency of administration of DuoNeb beyond these guidelines has not been studied and the safety and efficacy of extra doses of albuterol sulfate or ipratropium bromide in addition to the recommended doses of DuoNeb have not been studied.

The use of DuoNeb can be continued as medically indicated to control recurring bouts of bronchospasm. If a previously effective regimen fails to provide the usual relief, medical advice should be sought immediately, as this is often a sign of worsening COPD, which would require reassessment of therapy

A Pari-LC-Plus™ nebulizer (with face mask or mouthpiece) connected to a PRONEB™ compressor was used to deliver DuoNeb to each patient in one U.S. clinical study The safety and efficacy of DuoNeb delivered by other nebulizers and compressors have not been established

DuoNeb should be administered via jet nebulizer connected to an air compressor with an adequate air flow, equipped with a mouthpiece or suitable face mask

HOW SUPPLIED

DuoNeb is supplied as a 3-mL sterile solution for nebulization in sterile low-density polyethylene unit-dose vials. Cards of five vials are placed into a foil pouch Supplied in cartons as listed below

NDC 49502-672-30	30 vials per carton
NDC 49502-672-60	60 vials per carton

Store between 2°C and 25°C (36°F and 77°F). Protect from light.

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03-485-21(BRS) May 2001

1 Gross N, Tashkin D, Miller R, et al Inhalation by nebulization of albuterol-ipratropium combination (Dey combination) is superior to either agent alone in the treatment of chronic obstructive pulmonary disease. *Respiration.* 1998;65:354-362.

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We encourage you to provide us with information and contacts for other qualified vent camps. And will con- tinue to recognize clinicians who support these worthwhile endeavors. There is nothing more precious than to see these individuals enjoying life through fun activities with their peers.

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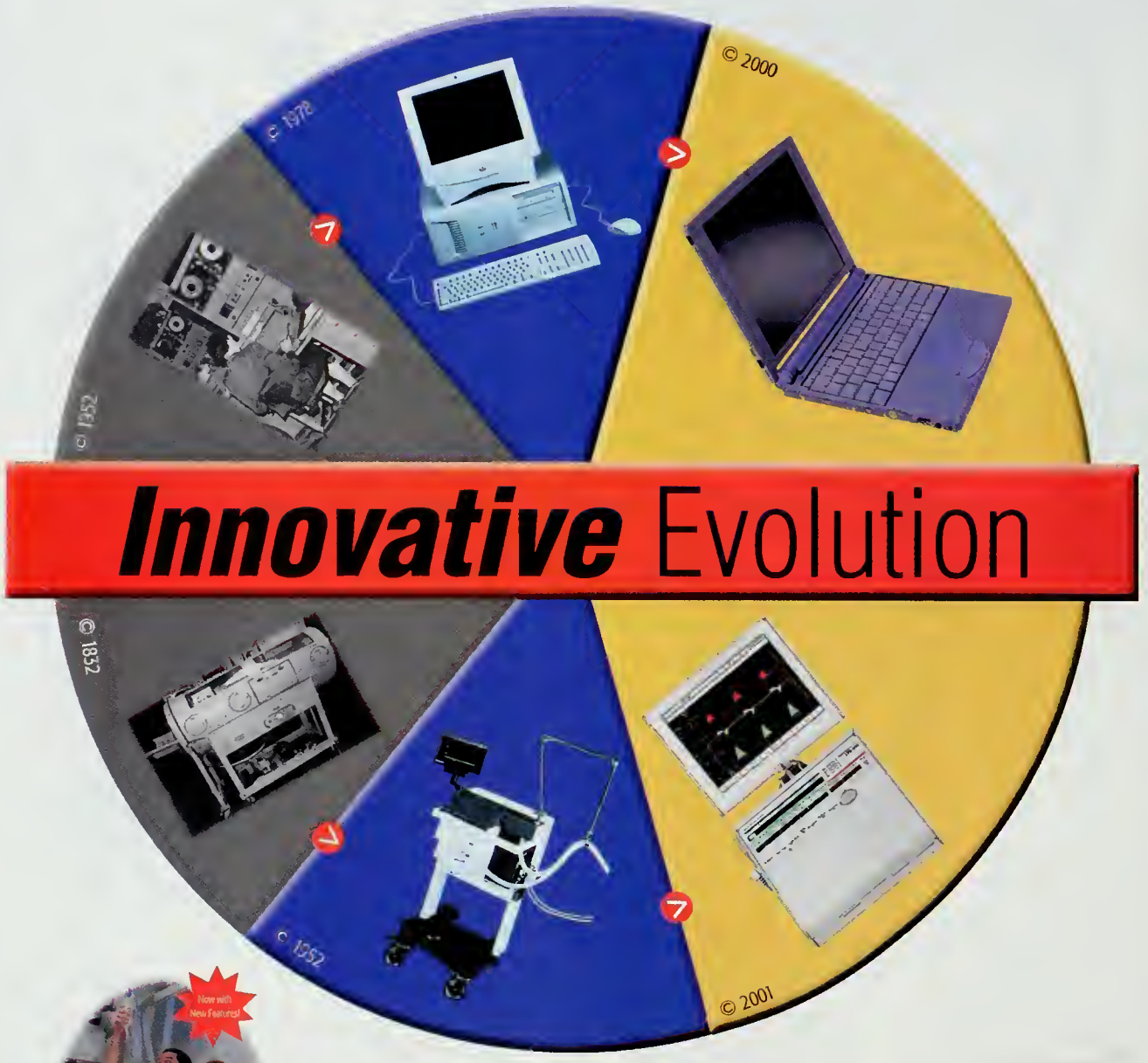

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- **Walt Disney World** is approximately 90 minutes from the downtown convention center... full of fun and excitement for the whole family.
- **Busch Gardens** is a 335-acre African-theme, family entertainment park offering naturalistic animal habitats, thrill rides, live entertainment, shopping, and dining. And just next door is Adventure Island with 37 acres of 17 water play areas and a beach volleyball complex.
- The **Florida Aquarium** features exhibits related to Florida's ecosystem and aquatic habitats. Highlights include: the Florida Wetlands Gallery, the Florida Bays and Beaches Gallery, and the Coral Reefs Gallery.
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Photos courtesy the Tampa Bay Convention and Visitors Bure

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Editorials, Commentaries, and Reviews to Note

Early and Aggressive Treatment Saves US Anthrax Victims—McCarthy M. Lancet 2001 Nov 17;358(9294):1703.

Flying and Cystic Fibrosis: Getting There and Back Safely—Webb AK. Thorax 2001 Nov;56(11):821-822.

Pulmonary Rehabilitation—British Thoracic Society Standards of Care Subcommittee on Pulmonary Rehabilitation. Thorax 2001 Nov;56(11):827-834.

Extracorporeal Membrane Oxygenation for Graft Failure After Heart Transplantation: Recent Vienna Experience—Taghavi S, Ankersmit HJ, Wieselthaler G, Gorlitzer M, Rajek A, Wolner E, Grimm M. J Thorac Cardiovasc Surg 2001 Oct;122(4):819-820.

Sleep-Disordered Breathing and Stroke: Is There a Rationale for Treatment? (editorial)—Parra O. Eur Respir J 2001 Oct;18(4):619-622.

Ethical Debate: The Distinction Between Withdrawing Life Sustaining Treatment Under the Influence of Paralysing Agents and Euthanasia: Are We Treading a Fine Line?—Street K, Henderson J. BMJ 2001 Aug 18;323(7309):388-389.

Ethical Debate: The Distinction Between Withdrawing Life Sustaining Treatment Under the Influence of Paralysing Agents and Euthanasia: The Parents Perspective on Withdrawing Treatment—Greig-Midlane H. BMJ 2001 Aug 18;323(7309):390.

Clinical Presentation of Inhalational Anthrax Following Bioterrorism Exposure: Report of 2 Surviving Patients—Mayer TA, Bersoff-Matcha S, Murphy C, Earls J, Harper S, Pauze D, et al. JAMA 2001 Nov 28;286(20):2549-2553.

The use of anthrax as a weapon of biological terrorism has moved from theory to reality in recent weeks. Following processing of a letter containing anthrax spores that had been mailed to a US senator, 5 cases of inhalational anthrax have occurred among postal workers employed at a major postal facility in Washington, DC. This report details the clinical presentation, diagnostic workup, and initial therapy of 2 of these patients. The clinical course is in some ways different from what has been described as the classic pattern for inhalational anthrax. One patient developed low-grade fever, chills, cough, and malaise 3 days prior to admission, and then progressive dyspnea and cough productive of blood-tinged sputum on the day of admission. The other patient developed progressively worsening headache of 3 days' duration, along with nausea, chills, and night sweats, but no respiratory symptoms, on the day of admission. Both patients had abnormal findings on chest radiographs. Non–contrast-enhanced computed tomography of the chest showing mediastinal adenopathy led to a presumptive diagnosis of inhalational anthrax in both cases. The diagnoses were confirmed by blood cultures and polymerase chain reaction testing. Treatment with antibiotics, including intravenous ciprofloxacin, rifampin, and clindamycin, and supportive therapy appears to have slowed the progression of inhalational anthrax and has resulted to date in survival.

Death Due to Bioterrorism-Related Inhalational Anthrax: Report of 2 Patients—Borio L, Frank D, Mani V, Chiriboga C, Pollanen M, Ripple M, et al. JAMA 2001 Nov 28;286(20):2554-2559.

On October 9, 2001, a letter containing anthrax spores was mailed from New Jersey to Washington, DC. The letter was processed at a major postal facility in Washington, DC, and opened in the Senate's Hart Office Building on October 15. Between October 19 and October 26, there were 5 cases of inhalational anthrax among postal workers who were employed at that major facility or who handled bulk mail originating from that facility. The cases of 2 postal workers who died of inhalational anthrax are reported here. Both patients had nonspecific prodromal illnesses. One patient developed predominantly gastrointestinal symptoms, including nausea, vomiting, and abdominal pain. The other patient had a "flulike" illness associated with myalgias and malaise. Both patients ultimately developed dyspnea, retrosternal chest pressure, and respiratory failure requiring mechanical ventilation. Leukocytosis and hemoconcentration were noted in both cases prior to death. Both patients had evidence of mediastinitis and extensive pulmonary infiltrates late in their course of illness. The durations of illness were 7 days and 5 days from onset of symptoms to death; both patients died within 24 hours of hospitalization. Without a clinician's high index of suspicion, the diagnosis of inhalational anthrax is difficult during nonspecific prodromal illness. Clinicians have an urgent need for prompt communication of vital epidemiologic information that could focus their diagnostic evaluation. Rapid diagnostic assays to distinguish more common infectious processes from agents of bioterrorism also could improve management strategies.

Comparison of the Lower Inflection Point on the Static Total Respiratory Compliance Curve with Outcomes in Postoperative Cardiothoracic Patients—Boyle M, Way P, Pinfold M, Lawrence J. Am J Crit Care 2001 Nov;10(6):399-407.

BACKGROUND: Routine use of positive end-expiratory pressure (based on the pressure at the lower inflection point on the static total respiratory compliance curve) along with a maneuver to recruit atelectatic lung has been advocated after cardiothoracic surgery. OBJECTIVES: To determine if the lower inflection point is related to outcomes in patients after sternotomy and cardiopulmonary bypass. METHOD: A prospective observational study involv-

ing estimation of the lower inflection point on the inflation pressure-volume plot obtained with a low-flow technique. Duration of intubation, length of stay, respiratory complications, and results of spirometry were compared between patients with a "high" inflection point (≥ 10 cm H_2O) and patients with a "low" inflection point (≤ 5 cm H_2O). RESULTS: Ninety-five patients were enrolled. After exclusion for incomplete data, 65 patients (49 men, 16 women; mean age, 66.1 years; SD, 9.5 years) were included. The mean lower inflection point was 6.33 cm H_2O (SD, 3.4 cm H_2O). A second lower inflection point was observed on 5 plots (mean, 21 cm H_2O; SD, 1.4 cm H_2O). Nine patients had high inflection points (mean, 13.1 cm H_2O; SD, 3.0 cm H_2O), and 33 had low inflection points (mean, 3.9 cm H_2O; SD, 0.98 cm H_2O). No outcome measures differed between groups. CONCLUSIONS: In patients with short intubation times and predictable postoperative course, general use of a lung recruitment strategy involving sustained inflations and adjustment of positive end-expiratory pressure based on the lower inflection point is difficult to justify.

End-Tidal Carbon Dioxide Measurements as a Prognostic Indicator of Outcome in Cardiac Arrest—Ahrens T, Schallom L, Bettorf K, Ellner S, Hurt G, O'Mara V, et al. Am J Crit Care 2001 Nov;10(6):391-398.

PURPOSE: To evaluate the use of end-tidal carbon dioxide values in predicting survival in cardiopulmonary arrest. BACKGROUND: The decision about when to terminate resuscitative efforts for patients with cardiopulmonary arrest is often subjective. End-tidal carbon dioxide values have been suggested as potential objective criteria for making this decision. METHODS: This study was a cooperative effort of the St Louis chapter of the American Association of Critical-Care Nurses and its members and involved 6 hospitals and an air evacuation service. All adult patients who had a cardiopulmonary arrest were eligiblefor the study. Once a patient with cardiac arrest was intubated, end-tidal carbon dioxide and cardiac rhythms were measured and recorded every 5 minutes for 20 minutes or until resuscitation efforts were terminated. Patients' survival at the time of the arrest, survival 24 hours after the arrest, and discharge status were followed up. RESULTS: A total of 127 patients were enrolled in the study. All but 1 patient with end-tidal carbon dioxide values less than 10 mm Hg died before discharge. End-tidal carbon dioxide values greater than 10 mm Hg were associated with various degrees of survival. Overall survival to discharge was less than 14%, regardless of the end-tidal carbon dioxide value. CONCLUSION: Measurements of end-tidal carbon dioxide can be used to accurately predict nonsurvival of patients with cardiopulmonary arrest. End-tidal carbon dioxide levels should be monitored during cardiopulmonary arrest and should be considered a useful prognostic value for determining the outcome of resuscitative efforts.

Predictors and Outcomes Associated with Early Extubation in Older Adults Undergoing Coronary Artery Bypass Surgery—Bezanson JL, Deaton C, Craver J, Jones E, Guyton RA, Weintraub WS. Am J Crit Care 2001 Nov;10(6):383-390.

BACKGROUND: Older age has been associated with prolonged mechanical ventilation after coronary artery bypass surgery. Prolonged mechanical ventilation contributes to increased morbidity and mortality and to use of limited financial resources among older adults. OBJECTIVES: To examine selected physiological and pathophysiological variables ofpresurgical-patients to predict duration of mechanical ventilation in older adults after coronary artery bypass surgery. METHODS: Nonrandomized study of a clinical database of 919 patients (≥ 65 years old) who had coronary artery bypass surgery between October 1996 and December 1997. RESULTS: Median elapsed time after coronary artery bypass surgery until extubation was used to sort patients into 2 groups: group 1, 6 hours or fewer (n = 464); and group 2, more than 6 hours (n = 455). With stepwise logistic regression, the physiological model included age (odds ratio, 1.05; p< 0.001) and female sex (odds ratio, 1.48; p = 0.005) with weak discrimination by group (concordance statistic = 0.5880). The pathophysiological model, which included renal insufficiency (odds ratio, 3.28; p = 0.01), previous peripheral vascular surgery (odds ratio, 2.87; p = 0.03), nonelective preoperative clinical status (odds ratio, 2.8; p = 0.006), congestive heart failure (odds ratio, 2.6; p < 0.001), and reoperation (odds ratio, 2.34; p = .007), showed moderate discrimination bygroup (concordance statistic = 0.6755). CONCLUSION: Many older adults were easily extubated and had good outcomes. The variables comorbid conditions and severity of illness provided better discrimination between extubation groups than a physiological model provided. Both predictive models allowed limited discrimination between groups.

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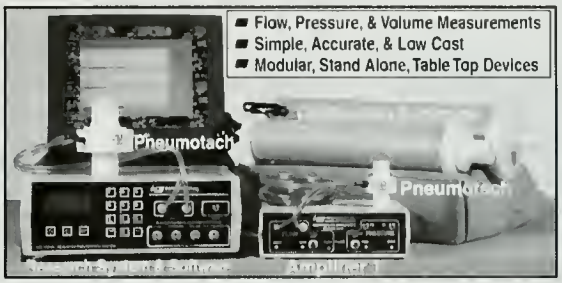

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Effect of Nurse-to-Patient Ratio in the Intensive Care Unit on Pulmonary Complications and Resource Use After Hepatectomy—Dimick JB, Swoboda SM, Pronovost PJ, Lipsett PA. Am J Crit Care 2001 Nov;10(6):376-382.

BACKGROUND: Nurse-to-patient ratios in the intensive care unit are associated with postoperative mortality, morbidity, and costs after some high-risk surgery. OBJECTIVE: To determine if having 1 nurse caring for 1 or 2 patients ("more nurses") versus 1 nurse caring for 3 or more patients ("fewer nurses") in the intensive care unit at night is associated with differences in clinical and economic outcomes after hepatectomy. METHODS: Statewide observational cohort study of 569 adults who had hepatic resection, 1994 to 1998. Hospital discharge data were linked to a prospective survey of organizational characteristics in the intensive care unit. Multivariate analysis was used to determine the association of nighttime nurse-to-patient ratios with in-hospital mortality, length of stay, hospital costs, and specific postoperative complications. RESULTS: A total of 240 patients at 25 hospitals had fewer nurses; 316 patients in 8 hospitals had more nurses. No significant asso-

ciation between nighttime nurse-to-patient ratios and in-hospital mortality was detected. The overall complication rate was 28%. By univariate analysis, patients with fewer nurses had increased risks for pulmonary failure (5.8% vs 1.6%, relative risk, 3.6; 95% CI, 1.3-10.1; p = 0.006) and reintubation (10.8% vs 1.9%, relative risk, 5.7; 95% CI, 2.4-13.7; p < 0.001). By multivariate analysis, patients with fewer nurses had increased risk for reintubation (odds ratio, 2.9; 95% CI, 1.0-8.1; p = 0.04) and a 14% increase (95% CI, 3%-23%; p = 0.007) or an additional $1248 (95% CI, $384-$2112; p = 0.005) in total hospital costs. CONCLUSIONS: Fewer nurses at night is associated with increased risk for specific postoperative pulmonary complications and with increased resource use in patients undergoing hepatectomy.

Comparison of Outcome Predictions Made by Physicians, by Nurses, and by Using the Mortality Prediction Model—Copeland-Fields L, Griffin T, Jenkins T, Buckley M, Wise LC. Am J Crit Care 2001 Sep;10(5):313-319.

BACKGROUND: Critical care nurses must collaborate with physicians, patients, and patients' families when making decisions about aggressiveness of care. However, few studies address nurses' ability to predict outcomes. OBJECTIVES: To compare predictions of survival outcomes made by nurses, by physicians, and by using the Mortality Prediction Model. METHODS: Predictions of survival and function and attitudes toward aggressiveness of care based on the predictions were recorded on questionnaires in the emergency department by emergency and intensive care unit physicians and by intensive care unit nurses at the time of admission to the unit between February and September 1995 for 235 consecutive adult nontrauma patients. Scores on the Mortality Prediction Model were calculated on admission. Data on 85 of the 235 patients were analyzed by using descriptive, χ^2, and correlational statistics. Nurses' predictions of function were compared with patients' actual outcomes 6 months after admission. RESULTS: Nurses' predictions of survival were comparable to those of emergency physicians and superior to those obtained by using the objective tool. Years of nursing experience had no relationship to attitudes toward aggressiveness of care. Nurses accurately predicted functional outcomes in 52% of the followed-up cases. Intensive care physicians were more accurate than nurses and emergency physicians in predicting survival. All predictions made by clinicians were superior to those obtained by using the model. CONCLUSIONS: Nurses can predict survival outcomes as accurately as physicians do. Greater sensitivity and specificity are necessary before clinical judgment or predictive tools can be considered as screens for determining aggressiveness of care.

End-of-Life Care in the Intensive Care Unit: A Challenge for Nurses—Miller PA, Forbes S, Boyle DK. Am J Crit Care 2001 Jul;10(4):230-237.

Results from several research studies combined with increasing public tensions surrounding physician-assisted suicide have fueled a growing awareness of the inadequacies of end-of-life care. Investigators also suggest that intensive care unit nurses have a limited role in end-of-life decision making and care planning. This article explores cultural issues influencing end-of-life care in intensive care units, explores factors surrounding the limited involvement of critical care nurses in end-of-life decision making and care planning, and offers recommendations for changing nursing practice. Because improving end-of-life care will require cultural changes, an understanding of the cultural issues involved is needed. Recommendations for changing nursing practice include a model of end-of-life care that incorporates the goals of both cure and comfort care, as well as a shared decision-making process. Nurses are essential to improving end-of-life care in today's intensive care units.

Elderly Patients' Reports of Physical Restraint Experiences in Intensive Care Units—Minnick A, Leipzig RM, Johnson ME. Am J Crit Care 2001 May;10(3):168-171.

BACKGROUND: Use of physical restraints has undesirable sequelae. As they weigh the risks and benefits of protocols for reducing the use of restraints, staff members in intensive care units, where restraints are most used in hospitals, need to know how well elderly patients remember being restrained and how patients perceive the use of restraints. OBJECTIVES: To estimate the proportion of patients who remember being restrained, describe the experience from the patients' perspectives, and describe any distress caused by use of restraints within the overall experience of being in the intensive care unit. METHODS: Transcripts of semistructured, audiotaped interviews of patients who had been in the medical or surgical intensive care unit in any of 3 eastern and midwestern medical centers were analyzed by question and for overall themes. RESULTS: Six patients (40%) remembered some aspect of being restrained but did not report great distress. Patients accepted restraints as needed because of the lack of alternatives. Patients reported remembering that they should not perform certain behaviors but being unable to stop themselves. Patients cited hallucinations and intubation as major stressors in the intensive care unit. Patients' continuing health problems after discharge from the intensive care unit severely limited recruitment of subjects. CONCLUSIONS: Patients do not remember great distress specifically related to the use of restraints, but the overall situation leading to use of restraints is disturbing if remembered. The discovery of methods to reduce the distress of intubation and hallucinations could decrease use of restraints.

A National Survey of Critical Care Nurses' Practices Related to Administration of Neuromuscular Blocking Agents—Foster JG, Kish SK, Keenan CH. Am J Crit Care 2001 May;10(3):139-145.

BACKGROUND: Recommendations on use of neuromuscular blocking agents include using peripheral nerve stimulators to monitor depth of blockade and concomitantly administering sedatives and/or analgesics. OBJECTIVE: To evaluate critical care nurses' practices in administering neuromuscular blocking agents. METHODS: A 16-item survey was mailed to 483 acute care facilities in the United States. Of these, 246 surveys (51%) were returned and analyzed to determine use of neuromuscular blocking agents, peripheral nerve stimulators, sedatives, and analgesics. Logistic regression analysis was used to find independent predictors of use of peripheral nerve stimulators. RESULTS: Seventy-five percent of respondents reported long-term use of neuromuscular blocking agents in critically ill patients. Of those, 63% monitored the level of blockade with peripheral nerve stimulators. Reasons for not using peripheral nerve stimulators included unavailability of equipment (48%), lack of training (36%), and insufficient evidence that peripheral nerve stimulators improve care (23%). Predictors of use of stimulators were facilities with more than 150 beds (p < 0.001) and administration of neuromuscular blocking agents by continuous infusion (p < 0.001). Ninety-five percent of respondents reported using concurrent analgesics/sedatives always or most of the time. Facilities with fewer than 10 beds in the intensive care unit used concurrent analgesics/sedatives significantly less often than did facilities with 10 beds or more (90% vs 98%, respectively; p = 0.03). CONCLUSIONS: Small and large facilities differ in concomitant use of analgesics/sedatives and peripheral nerve stimulators. Education and research are needed to ensure that patients receive adequate monitoring and sedation during administration of neuromuscular blocking agents.

Quiet Time: A Nursing Intervention to Promote Sleep in Neurocritical Care Units—Olson DM, Borel CO, Laskowitz DT, Moore DT, McConnell ES. Am J Crit Care 2001 Mar;10(2):74-78.

BACKGROUND: Patients in intensive care units are often sleep deprived, yet little research exists on the impact of nursing care on promoting sleep. OBJECTIVES: To determine if implementing a "quiet time" protocol to reduce external environmental stimuli is associated with increased frequency of sleep among patients in a neurocritical care unit. METHODS: Patients were observed 8 times each day before and after implementation of a protocol in which environmental sounds and lights were decreased from 2 AM to 4 AM and from 2 PM to 4 PM. Data collected at 2:45 AM, 3:30 AM, 2:45 PM, and 3:30 PM on patients with scores of 10 or greater on the Glasgow Coma Scale were

analyzed. A total of 2975 observations were made on a total of 239 patients: 1446 observations on 118 patients in the control group and 1529 observations on 121 patients in the intervention group. RESULTS: The percentage of patients observed asleep was significantly higher during the months the quite-time period was implemented than during the control period before the intervention was started. The increase in sleep behavior was associated with decreased sound and light levels achieved during the quiet time. Patients observed during the intervention period were 1.6 times more likely to be asleep during the quiet time than were patients observed during the control period ($p < 0.001$). CONCLUSIONS: A concentrated effort by staff to reduce environmental stimuli at discrete preset intervals increases the likelihood of sleep during scheduled quiet time in the neurocritical care unit.

Long-Term Effects of Spontaneous Breathing During Ventilatory Support in Patients with Acute Lung Injury—Putensen C, Zech S, Wrigge H, Zinserling J, Stuber F, Von Spiegel T, Mutz N. Am J Respir Crit Care Med 2001 Jul 1;164(1):43-49.

Improved gas exchange has been observed during spontaneous breathing with airway pressure release ventilation (APRV) as compared with controlled mechanical ventilation. This study was designed to determine whether use of APRV with spontaneous breathing as a primary ventilatory support modality better prevents deterioration of cardiopulmonary function than does initial controlled mechanical ventilation in patients at risk for acute respiratory distress syndrome (ARDS). Thirty patients with multiple trauma were randomly assigned to either breathe spontaneously with APRV (APRV Group) ($n = 15$) or to receive pressure-controlled, time-cycled mechanical ventilation (PCV) for 72 h followed by weaning with APRV (PCV Group) ($n = 15$). Patients maintained spontaneous breathing during APRV with continuous infusion of sufentanil and midazolam (Ramsay sedation score [RSS] of 3). Absence of spontaneous breathing (PCV Group) was induced with sufentanil and midazolam (RSS of 5) and neuromuscular blockade. Primary use of APRV was associated with increases ($p < 0.05$) in respiratory system compliance (CRS), arterial oxygen tension (P_{aO_2}), cardiac index (CI), and oxygen delivery (D_{O_2}), and with reductions ($p < 0.05$) in venous admixture ($\dot{Q}_{VA}/\dot{Q}_T$), and oxygen extraction. In contrast, patients who received 72 h of PCV had lower CRS, P_{aO_2}, CI, D_{O_2}, and $\dot{Q}_{VA}/\dot{Q}_T$ values ($p < 0.05$) and required higher doses of sufentanil ($p < 0.05$), midazolam ($p < 0.05$), noradrenalin ($p < 0.05$), and dobutamine ($p < 0.05$). CRS, P_{aO_2}), CI and D_{O_2} were lowest ($p < 0.05$) and $\dot{Q}_{VA}/\dot{Q}_T$ was highest ($p < 0.05$) during PCV. Primary use of APRV was consistently associated with a shorter duration of ventilatory support (APRV Group: 15 ± 2 d [mean ± SEM]; PCV Group: 21 ± 2 d) ($p < 0.05$) and length of intensive care unit (ICU) stay (APRV Group: 23 ± 2 d; PCV Group: 30 ± 2 d) ($p < 0.05$). These findings indicate that maintaining spontaneous breathing during APRV requires less sedation and improves cardiopulmonary function, presumably by recruiting nonventilated lung units, requiring a shorter duration of ventilatory support and ICU stay.

The Effect of Nebulized Epinephrine on Respiratory Mechanics and Gas Exchange in Bronchiolitis—Numa AH, Williams GD, Dakin CJ. Am J Respir Crit Care Med 2001 Jul 1;164(1):86-91.

Nebulized epinephrine has been advocated as a treatment for airway obstruction in infants with bronchiolitis; however, its effect on respiratory mechanics and gas exchange has been poorly documented to date. We performed a preinterventional and postinterventional study with primary outcome measures of mechanics (measured by single-breath occlusion passive deflation) and oxygenation and ventilation indices in order to measure the effects of nebulized epinephrine in infants requiring mechanical ventilation for RSV-positive bronchiolitis. A two-compartment model was used to describe respiratory mechanics in patients with nonlinear flow-volume curves. Nebulized epinephrine (0.5 mg/kg) was administered to 15 patients (median age, 0.19 yr; weight, 4.4 kg). Resistance decreased significantly in slow and fast compartments in 87 and 70% of patients, respectively. Median resistance

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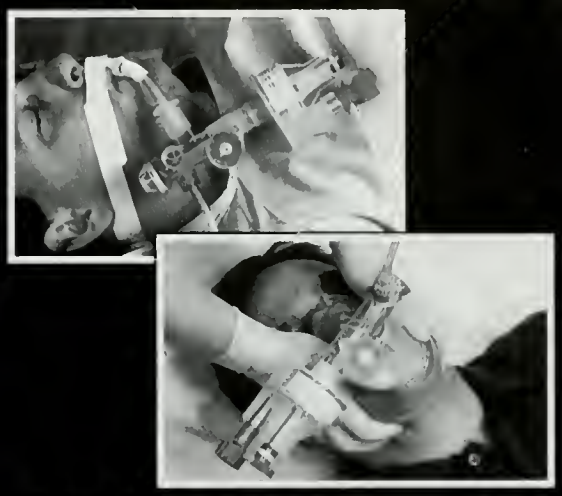

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in the slow compartment decreased from 0.427 to 0.198 cm H_2O/mL/s ($p = 0.0015$) and in the fast compartment from 0.167 to 0.116 cm H_2O/ mL/s ($p = 0.018$). Compliance, oxygenation index, and ventilation index were not significantly changed after administration of epinephrine. We conclude that nebulized epinephrine substantially improves respiratory system resistance but not oxygenation or ventilation indices. This may be because of the effects of epinephrine on oxygen consumption or ventilation-perfusion matching.

Recruitment and Derecruitment During Acute Respiratory Failure: An Experimental Study—Pelosi P, Goldner M, McKibben A, Adams A, Eccher G, Caironi P, et al. Am J Respir Crit Care Med 2001 Jul 1;164(1):122-130.

We aimed to elucidate the relationships between pleural (Ppl), esophageal (Pes), and superimposed gravitational pressures in acute lung injury, and to understand the mechanisms of recruitment and derecruitment. In six dogs with oleic acid respiratory failure, we measured Pes and Ppl in the uppermost, middle, and most dependent lung regions. Each dog was studied at positive end-expiratory pressure (PEEP) of 5 and 15 cm H_2O and three levels of tidal volume (V_T; low, medium, and high). For each PEEP-V_T combination, we obtained a computed tomographic (CT) scan at end-inspiration and end-expiration. The variations of Ppl and Pes pressures were correlated ($r = 0.86 \pm 0.07$, $p < 0.0001$), as was the vertical gradient of transpulmonary (PL) and superimposed pressure ($r = 0.92$, $p < 0.0001$). Recruitment proceeded continuously along the entire volume-pressure curve. Estimated threshold opening pressures were normally distributed (mode = 20 to 25 cm H_2O). The amount of end-expiratory collapse at the same PEEP and PL was significantly lower when ventilation was performed at high V_T. End-inspiratory and end-expiratory collapse were highly correlated ($r = 0.86$, $p < 0.0001$), suggesting that as more tissue is recruited at end-inspiration, more remains recruited at end-expiration. When superimposed pressure exceeded applied airway pressure (Paw), collapse significantly increased.

Recruitment and Derecruitment During Acute Respiratory Failure: A Clinical Study—Crotti S, Mascheroni D, Caironi P, Pelosi P, Ronzoni G, Mondino M, et al. Am J Respir Crit Care Med 2001 Jul 1;164(1):131-140.

In a model of acute lung injury, we showed that positive end-expiratory pressure (PEEP) and tidal volume (V_T) are interactive variables that determine the extent of lung recruitment, that recruitment occurs across the entire range of total lung capacity, and that superimposed pressure is a key determinant of lung collapse. Aiming to verify if the same rules apply in a clinical setting, we randomly ventilated five ALI/ARDS patients with 10, 15, 20, 30, 35, and 45 cm H_2O plateau pressure and 5, 10, 15, and 20 cm H_2O of PEEP. For each PEEP-V_T condition, we obtained computed tomography at end inspiration and end expiration. We found that recruitment occurred along the entire volume-pressure curve, independent of lower and upper inflection points, and that estimated threshold opening pressures were normally distributed (mode = 20 cm H_2O). Recruitment occurred progressively from nondependent to dependent lung regions. Overstretching was not associated with hyperinflation. Derecruitment did not parallel deflation, and estimated threshold closing pressures were normally distributed (mode = 5 cm H_2O). End-inspiratory and end-expiratory collapse were correlated, suggesting a plateau-PEEP interaction. When superimposed gravitational pressure exceeded PEEP, end-expiratory collapse increased. We concluded that the rules governing recruitment and derecruitment equally apply in an oleic acid model and in human ALI/ARDS.

Does This Patient Have Clubbing?—Myers KA, Farquhar DR. JAMA 2001 Jul 18;286(3):341-347.

CONTEXT: The association between digital clubbing and a host of diseases has been recognized since the time of Hippocrates. Although the features of advanced clubbing are familiar to most clinicians, the presence of early clubbing is often a source of debate. OBJECTIVE: To perform a systematic review of the literature for information on the precision and accuracy of clinical examination for clubbing. DATA SOURCES: The MEDLINE database from January 1966 to April 1999 was searched for English-language articles related to clubbing. Bibliographies of all retrieved articles and of standard textbooks of physical diagnosis were also searched. STUDY SELECTION: Studies selected for data extraction were those in which quantitative or qualitative assessment for clubbing was described in a series of patients. Sixteen studies met these criteria and were included in the final analysis. DATA EXTRACTION: Data were extracted by both authors, who independently reviewed and appraised the quality of each article. Data extracted included quantitative indices for distinguishing clubbed from normal digits, precision of clinical examination for clubbing, and accuracy of clubbing as a marker of selected diseases. DATA SYNTHESIS: The profile angle, hyponychial angle, and phalangeal depth ratio can be used as quantitative indices to assist in identifying clubbing. In individuals without clubbing, values for these indices do not exceed 176 degrees, 192 degrees, and 1.0, respectively. When clinicians make a global assessment of clubbing at the bedside, interobserver agreement is variable, with kappa values ranging between 0.39 and 0.90. Because of the lack of an objective diagnostic criterion standard, accuracy of physical examination for clubbing is difficult to determine. The accuracy of clubbing as a marker of specific underlying disease has been determined for lung cancer (likelihood ratio, 3.9 with phalangeal depth ratio in excess of 1.0) and for inflammatory bowel disease (likelihood ratio, 2.8 and 3.7 for active Crohn disease and ulcerative colitis, respectively, if clubbing is present). CONCLUSIONS: We recommend use of the profile angle and phalangeal depth ratio as quantitative indices in identifying clubbing. Clinical judgment must be exercised in determining the extent of further evaluation for underlying disease when these values exceed 180 degrees and 1.0, respectively.

Spirometry in Children Aged 3 To 5 Years: Reliability of Forced Expiratory Maneuvers—Crenesse D, Berlioz M, Bourrier T, Albertini M. Pediatr Pulmonol 2001 Jul;32(1):56-61.

The aim of this study was to evaluate the feasibility and reproducibility of forced expiratory maneuvers during standard spirometric evaluation in preschool children. Among 570 young children attending our laboratory, we retrospectively selected 355 patients (14% 3-4-year-olds, 48% 4-5-year-olds, and 38% 5-6-year-olds) who carried out spirometric tests for the first time. The indications for such tests were history of asthma (70%), followed by chronic cough (20%) and other miscellaneous conditions (10%). Eighty-eight, 175, and 92 children performed one, two, and three acceptable tests respectively. Forced expired volume in 1 sec (FEV_1) and forced vital capacity (FVC) did not differ significantly between attempts in children performing either two or three attempts. Forced expiratory time (FET), i.e., the total time required for the forced expiratory maneuver, was 1.7 ± 0.1 sec (mean ± SEM), and was no greater than 1 sec in 21.3% of all tested children. Consequently, FEV_1 does not appear to be well-suited to this age group. Forced expiratory volume in 0.50 and 0.75 sec ($FEV_{0.5}$, $FEV_{0.75}$) were thus measured in the group of children performing three attempts ($n = 92$), and there was no statistical difference between attempts. In 267 children performing two or three tests, the ATS criteria of reproducing FEV_1 and FVC within ≤ 0.1 L seemed to be preferable in this young population. Indeed, more than 70% of the tested children presented their two best efforts (FVC and FEV_1) not varying by more than 0.1 L. Individual coefficients of variation (CV = SD/mean x 100%) over three tests for FEV_1 and FVC were $6.71 \pm 0.53\%$ and $6.35 \pm 0.41\%$ (mean ± SEM), respectively. These results show that forced expiratory tests are not always feasible in young children, but that 55% (196/355) of our selected population performed reliable maneuvers (at least two FVC and FEV_1 reproducible within 0.1 L), provided that they were supervised by a carefully trained pediatric medical staff.

Work of Breathing Associated with Pressure Support Ventilation in Two Different Ventilators—Sanders RC Jr, Thurman TL, Holt SJ, Taft K, Heulitt MJ. Pediatr Pulmonol 2001 Jul;32(1):62-70.

The purpose of this study was to compare the work of breathing during pressure support ventilation (PSV) with positive end expiratory pressure (PEEP)

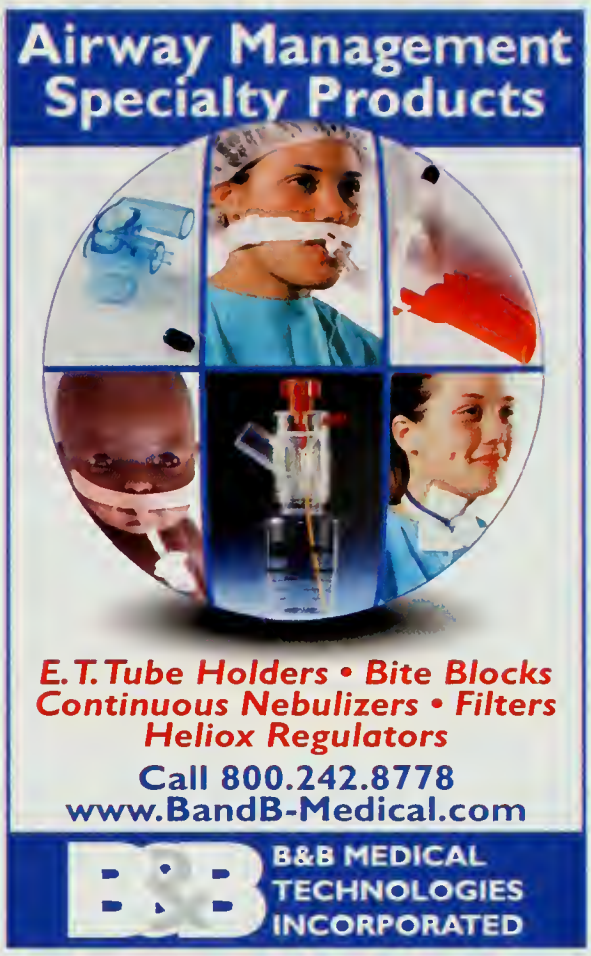
utilizing the Siemens SV300 (SV300) and Dräger Evita 4 (EV4) ventilators. Our hypothesis was that patients' work of breathing (WOB_P) would be unchanged in PSV utilizing flow triggering (FT) in both the SV300 and EV4. We compared two ventilators using six healthy, intubated, sedated, spontaneously breathing pigs weighing approximately 10 kg each. WOB_P (j/L) and ventilator work of breathing (WOB_V) (j/L) were measured using a portable monitor which utilizes an esophageal balloon and flow transducer. Each breath was further analyzed for duration of inspiratory effort and negative deflection of pressure needed to trigger PSV. Animals were studied with the SV300 and EV4 on a pressure support of 5 cm H_2O and PEEP settings of 0 and 5 cm H_2O. Data were analyzed using the Wilcoxon signed rank test with significance set at $p \leq 0.05$ WOB_P was 90% (PS 5, PEEP 0) and 52% (PS 5, PEEP 5) lower on the SV300 compared to the EV4. WOB_V was 94% (PS 5, PEEP 0) and 39% (PS 5, PEEP 5) higher on the SV300 when compared to the EV4. The change in airway pressure (Δp) from baseline and most negative deflection of pressure were greater with the EV4 as compared to the SV300, although Δ pressure was not found to be statistically significant on PS of 5 cm H_2O and PEEP of 5 cm H_2O. The SV300 also had shorter duration of inspiratory effort from initiation of breath to most negative deflection of pressure and to maximum flow than the EV4. In conclusion, these results suggest there are significant differences in WOB_P between the SV300 and EV4 ventilators. Response time of the ventilators may explain the differences in duration of inspiratory effort and the patient's work of breathing and thus may have an impact on weaning time for ventilated patients.

Patient-Ventilator Interactions in New Modes of Patient-Triggered Ventilation—Abubakar KM, Keszler M. Pediatr Pulmonol 2001 Jul;32(1):71-75.

Recently, synchronized modes of conventional mechanical ventilation became available for neonatal ventilatory support, but there has been little information regarding details of patient-ventilator interactions during pressure support, volume support, or any other volume-targeted modes of synchronized ventilation in newborn infants. Our objective was to obtain comparative data on patient-ventilator interactions and stability of delivered tidal volume (V_T) for the different modes of synchronized mechanical ventilation in stable ventilated newborn infants. We examined the effects of pressure support ventilation (PSV) and volume guarantee (VG) modes of a prototype Dräger Babylog ventilator on peak and mean airway pressures (PIP and Paw), inspiratory time (t_{in}), and V_T in 23 ventilated newborn infants. Twelve infants were studied while on assist/control (AC) and 11 on synchronized intermittent mandatory ventilation (SIMV). Mean birth weight was 1,650 ± 1,180 g, gestational age 31 ± 6 weeks, and age at time of study was 19 ± 26 days. Data for 400-600 breaths from each infant were downloaded directly from the ventilator pressure and volume-monitoring module, and analyzed using ANOVA for repeated measures. Mean values and breath-to-breath variability were compared for 20-min periods of AC or SIMV followed by PSV, PSV+VG, and back to baseline AC or SIMV. PSV and PSV+VG led to shorter tin and thus to lower Paw, compared to AC. Mean PIP was similar across all AC modes but more variable during VG, reflecting the servocontrol of PIP. V_T did not differ between AC modes, but was significantly less variable with VG added. PSV and PSV+VG led to lower and less variable PIP and Paw, compared to SIMV, because tin was shorter and every breath was supported in PSV and PSV+VG. V_T was similar in SIMV, PSV, and PSV+VG, but less variable with PSV+VG. Arterial blood gas tensions were similar across all ventilation modes. We conclude that the ventilator prototype functioned as intended. Breath-to-breath tidal volume variability was significantly reduced in VG modes, although not completely eliminated.

Interventions for Smoking Cessation in Hospitalised Patients: A Systematic Review—Munafo M, Rigotti N, Lancaster T, Stead L, Murphy M. Thorax 2001 Aug;56(8):656-663.

BACKGROUND: An admission to hospital provides an opportunity to help people stop smoking. Individuals may be more open to help at a time of perceived vulnerability, and may find it easier to quit in an environment where smoking is restricted or prohibited. Providing smoking cessation services during hospitalisation may help more people to attempt and sustain an attempt to quit. The purpose of this paper is to systematically review the effectiveness of interventions for smoking cessation in hospitalised patients. METHODS: We searched the Cochrane Tobacco Addiction Group register, CINAHL, and the Smoking and Health database for studies of interventions for smoking cessation in hospitalised patients. Randomised and quasi-randomised trials of behavioural, pharmacological, or multi-component interventions to help patients stop smoking conducted with hospitalised patients who were current smokers or recent quitters were included. Studies of patients admitted for psychiatric disorders or substance abuse, those that did not report abstinence rates, and those with follow up of less than 6 months were excluded. Two of the authors extracted data independently for each paper, with assistance from others. RESULTS: Intensive intervention (inpatient contact plus follow up for at least 1 month) was associated with a significantly higher cessation rate compared with controls (Peto odds ratio (OR) 1.82, 95% CI 1.49 to 2.22). Any contact during hospitalisation followed by minimal follow up failed to detect a statistically significant effect on cessation rate, but did not rule out a 30% increase in smoking cessation (Peto OR 1.09, 95% CI 0.91 to 1.31). There was insufficient evidence to judge the effect of interventions delivered only during the hospital stay. Although the interventions increased quit rates irrespective of whether nicotine replacement therapy (NRT) was used, the results for NRT were compatible with other data indicating that it increases quit rates. There was no strong evidence that clinical diag-



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nosis affected the likelihood of quitting. CONCLUSIONS: High intensity behavioural interventions that include at least 1 month of follow up contact are effective in promoting smoking cessation in hospitalised patients.

The Voluntary Drive to Breathe Is Not Decreased in Hypercapnic Patients with Severe COPD—Topeli A, Laghi F, Tobin MJ. Eur Respir J 2001 Jul;18(1):53-60.

How do the respiratory centres of patients with chronic obstructive pulmonary disease (COPD) and hypercapnia respond to acute increases in inspiratory load? A depressed respiratory motor output has long been postulated, but studies on this issue have yielded inconsistent results, partly due to limitations of investigative techniques. Many of these limitations can be overcome by the twitch interpolation technique, which is capable of accurately quantifying the degree of diaphragmatic activation, termed the voluntary drive to breathe. The hypothesis that patients with COPD and hypercapnia compensate for an acute increase in mechanical load on the inspiratory muscles with a lower voluntary drive to breathe than is the case with normocapnic patients was tested. Measurements were obtained in 15 patients with COPD, six of whom displayed hypercapnia and nine normocapnia. The maximum degree of diaphragmatic activation, expressed as a voluntary activation index (mean ± SEM), was higher in hypercapnic than in normocapnic patients (98.7 ± 0.7 versus 94.5 ± 0.9% ($p = 0.006$)), as was the mean value (94.5 ± 0.7 versus 88.5 ± 1.9% ($p = 0.01$)). Within-patient values of the index were also less variable in the hypercapnic patients (coefficients of variation, 3.4 ± 0.3 versus 6.1 ± 0.9%, $p = 0.01$). Multiple regression analysis revealed the ratio of dynamic elastance to maximum transdiaphragmatic pressure, an index of inspiratory muscle loading, and pH as the only variables that correlated with maximum voluntary activation index ($r^2 = 0.69$, $p = 0.02$ for each variable). Contrary to the hypothesis, it was concluded that voluntary activation of the diaphragm was greater and less variable in hypercapnic patients than normocapnic patients with severe chronic obstructive pulmonary disease during an acute increase in inspiratory mechanical load. Whether greater diaphragmatic recruitment during episodes of a severe exacerbation of chronic obstructive pulmonary disease provides a survival advantage for hypercapnic patients with chronic obstructive pulmonary disease remains to be determined.

Dose-Response Effect of Oxygen on Hyperinflation and Exercise Endurance in Nonhypoxaemic COPD Patients—Somfay A, Porszasz J, Lee SM, Casaburi R. Eur Respir J 2001 Jul;18(1):77-84.

Dynamic hyperinflation contributes to exertional breathlessness and reduced exercise tolerance in chronic obstructive pulmonary disease (COPD) patients. This study examined whether oxygen supplementation results in a dose-dependent decrease in hyperinflation associated with functional and symptomatic improvement. Ten severe COPD patients without clinically significant oxygen (O_2) desaturation during exercise, and seven healthy subjects, performed five exercise tests at 75% of maximally tolerated work rate. Inspired oxygen fraction (F_{IO_2}) was varied (0.21, 0.3, 0.5, 0.75 and 1.0) among tests in a randomized order. Ventilation ($\dot{V}_E$) was measured, and end-inspiratory (EILV) and end-expiratory (EELV) lung volume changes were assessed from inspiratory capacity manoeuvres. In the patients, compared to room air, endurance time increased with $F_{IO_2} = 0.3$ (mean ± SEM 92 ± 20%) and increased further with $F_{IO_2} = 0.5$ (157 ± 30%). At isotime, compared to room air, there were significant reductions in dyspnoea score, EELV, EILV, $\dot{V}_E$ and respiratory frequency (fR) with $F_{IO_2} = 0.3$. Improved endurance time negatively correlated with change in EELV ($r = 0.48$, $p = 0.002$) and EILV ($r = 0.43$, $p = 0.005$). The dyspnoea rating decrease correlated with fR decrease. In healthy subjects, smaller $\dot{V}_E$ and fR decreases were observed at $F_{IO_2} = 0.5$, accompanied by more modestly increased endurance. Oxygen supplementation during exercise induced dose-dependent improvement in endurance and symptom perception in nonhypoxaemic chronic obstructive pulmonary disease patients, which may be partly related to decreased hyperinflation and slower breathing pattern. This effect is maximized at an inspired oxygen fraction of 0.5.

The Effects of Pressurization Rate on Breathing Pattern, Work of Breathing, Gas Exchange and Patient Comfort in Pressure Support Ventilation—Chiumello D, Pelosi P, Croci M, Bigatello LM, Gattinoni L. Eur Respir J 2001 Jul;18(1):107-114.

The aim of this study was to investigate the effects of different pressurization rates during pressure support ventilation on breathing pattern, work of breathing, gas exchange and patient comfort in patients with acute lung injury. The pressurization rate modifies the initial pressure ramp by changing the initial peak flow rate: the increase in pressurization rate is associated with a decrease in the time to reach the level of pressure support ventilation by increasing the peak flow rate. Ten intubated patients (age 64±17 yrs, body mass index 24±17 Kg x m^{-2}, arterial oxygen tension/inspired oxygen fraction 214±59) were studied in random order varying the pressurization rate at 5 and 15 cm H_2O of pressure support ventilation. Breathing comfort was evaluated by a visual analogue scale. Increasing the pressurization rate caused an increase of peak flow rate from 473±141 mL x s^{-1} to 758±302 mL x s^{-1} at pressure support ventilation 5 ($p < 0.05$) and from 481±126 mL x s^{-1} to 1,121±175 mL x s^{-1} at pressure support ventilation 15 ($p < 0.05$). At the lowest pressurization rate the tidal volume was the lowest, the respiratory rate and the work of breathing were the highest ($p < 0.05$) compared with other pressurization rates. Excluding the lowest pressurization rate, in all the other pressurization rates tested the breathing pattern and the work of breathing did not change. The lowest and the highest pressurization rates caused the worst patient comfort ($p < 0.05$). The gas exchange was stable throughout the study. The presented results suggest: 1) the lowest pressurization rate caused the lowest tidal volume, highest respiratory rate and highest work of breathing; 2) at the other pressurization rates no differences in breathing pattern and work of breathing were observed; and 3) the patient's comfort was worse at the lowest and highest pressurization rates.

Noninvasive Mechanical Ventilation in Acute Hypoxaemic Respiratory Failure—Wysocki M, Antonelli M. Eur Respir J 2001 Jul;18(1):209-220.

In acute hypoxaemic respiratory failure (HRF), oxygenation, reduction in the work of breathing and in dyspnoea may be achieved by delivering noninvasive mechanical ventilation (NIMV). Several uncontrolled and 13 randomized controlled studies (RCS) were reviewed. Uncontrolled studies confirmed the feasibility and the possibility to improve arterial blood oxygenation with NIMV. The 13 RCS compared NIMV versus a conventional approach in a total of 720 patients with HRF. Endotracheal intubation was required in 186 of the 358 patients (median (95% confidence interval (CI)) 51%, (40-63%)) assigned to a conventional approach and in 107 of the 362 patients (29% (20-39%)) assigned to NIMV. Eleven of the 13 RCS found a reduction in the rate of endotracheal intubation with NIMV with an absolute risk reduction of 31% (30-33%). Ten of the 13 RCS found a reduction in the mortality rate which was 30% (19-40%) in the control group and 19% (13-26%) in the NIMV group. The mean absolute risk reduction was 15% (10-20%). In conclusion, noninvasive ventilation appears to be a useful method in avoiding endotracheal intubation and probably in reducing the morbidity of patients with hypoxaemic respiratory failure.

Prospective Trial of High-Frequency Oscillation in Adults with Acute Respiratory Distress Syndrome—Mehta S, Lapinsky SE, Hallett DC, Merker D, Groll RJ, Cooper AB, et al. Crit Care Med 2001 Jul;29(7):1360-1369.

OBJECTIVE: To evaluate the safety and efficacy of high-frequency oscillatory ventilation (HFOV) in adult patients with the acute respiratory distress syndrome (ARDS) and oxygenation failure. DESIGN: Prospective, clinical study. SETTING: Intensive care and burn units of two university teaching hospitals. PATIENTS: Twenty-four adults (10 females, 14 males, aged 48.5 ± 15.2 yrs, Acute Physiology and Chronic Health Evaluation II score 21.5 ± 6.9) with ARDS (lung injury score 3.4 ± 0.6, P_{aO_2}/F_{IO_2} 98.8 ± 39.0 mm Hg, and oxygenation index 32.5 ± 19.6) who met one of the following cri-

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teria: $P_{aO_2} \leq 65$ mm Hg with $F_{IO_2} \geq 0.6$, or plateau pressure ≥ 35 cm H_2O. INTERVENTIONS: HFOV was initiated in patients with ARDS after varying periods of conventional ventilation (CV). Mean airway pressure (Paw) was initially set 5 cm H_2O greater than Paw during CV, and was subsequently titrated to maintain oxygen saturation between 88% and 93% and $F_{IO_2} \leq 0.60$. MEASUREMENTS AND MAIN RESULTS: FIO2, Paw, pressure amplitude of oscillation, frequency, blood pressure, heart rate, and arterial blood gases were monitored during the transition from CV to HFOV, and every 8 hrs thereafter for 72 hrs. In 16 patients who had pulmonary artery catheters in place, cardiac hemodynamics were recorded at the same time intervals. Throughout the HFOV trial, Paw was significantly higher than that applied during CV. Within 8 hrs of HFOV application, and for the duration of the trial, F_{IO_2} and P_{aCO_2} were lower, and P_{aO_2}/F_{IO_2} was higher than baseline values during CV. Significant changes in hemodynamic variables following HFOV initiation included an increase in pulmonary artery occlusion pressure (at 8 and 40 hrs) and central venous pressure (at 16 and 40 hrs), and a reduction in cardiac output throughout the course of the study. There were no significant changes in systemic or pulmonary pressure associated with initiation and maintenance of HFOV. Complications occurring during HFOV included pneumothorax in two patients and desiccation of secretions in one patient. Survival at 30 days was 33%, with survivors having been mechanically ventilated for fewer days before institution of HFOV compared with nonsurvivors (1.6 ± 1.2 vs. 7.8 ± 5.8 days; $p = 0.001$). CONCLUSIONS: These findings suggest that HFOV has beneficial effects on oxygenation and ventilation, and may be a safe and effective rescue therapy for patients with severe oxygenation failure. In addition, early institution of HFOV may be advantageous.

Lower Inflection Point and Recruitment with PEEP in Ventilated Patients with Acute Respiratory Failure—Mergoni M, Volpi A, Bricchi C, Rossi A. J Appl Physiol 2001 Jul;91(1):441-450.

The lower inflection point (LIP) on the total respiratory system pressure-volume (P-V) curve is widely used to set positive end-expiratory pressure (PEEP) in patients with acute respiratory failure (ARF) on the assumption that LIP represents alveolar recruitment. The aims of this work were to study the relationship between LIP and recruited volume (RV) and to propose a simple method to quantify the RV. In 23 patients with ARF, respiratory system P-V curves were obtained by means of both constant-flow and rapid occlusion technique at four different levels of PEEP and were superimposed on the same P-V plot. The RV was measured as the volume difference at a pressure of 20 cm H_2O. A third measurement of the RV was done by comparing the exhaled volumes after the same distending pressure of 20 cm H_2O was applied (equal pressure method). RV increased with PEEP ($p < 0.0001$); the equal pressure method compares favorably with the other methods ($P = 0.0001$ by correlation), although individual data cannot be superimposed. No significant difference was found when RV was compared with PEEP in the group of patients with a LIP ≤ 5 cm H_2O and the group with a LIP > 5 cm H_2O (76.9 ± 94.3 vs. 61.2 ± 51.3, 267.7 ± 109.9 vs. 209.6 ± 73.9, and 428.2 ± 216.3 vs. 375.8 ± 145.3 mL with PEEP of 5, 10, and 15 cm H_2O, respectively). A RV was found even when a LIP was not present. We conclude that the recruitment phenomenon is not closely related to the presence of a LIP and that a simple method can be used to measure RV.

Lung Function 5 Yr After Lung Volume Reduction Surgery for Emphysema—Gelb AF, McKenna RJ Jr, Brenner M, Epstein JD, Zamel N. Am J Respir Crit Care Med 2001 Jun;163(7):1562-1566.

Current datum more than 2 yr after lung volume reduction surgery (LVRS) for emphysema is limited. This prospective study evaluates pre-LVRS baseline and 5-yr results in 26 symptomatic patients (mean age 67 ± 6 yr) (mean ± SD) who underwent bilateral, targeted upper lobe stapled LVRS using video-assisted thoracoscopy. Baseline forced expiratory volume in 1 s (FEV_1) was 0.7 ± 0.2 L (mean ± SD), 29 ± 10% predicted. Following LVRS, with none lost to follow-up, mortality due to respiratory failure at 0.5, 1, 2, 3, 4, and 5 yr was 4%, 4%, 19%, 31%, 46%, and 58%, respectively. Increase above baseline for $FEV_1 > 200$ mL and/or FVC > 400 mL at 1, 2, 3, 4, and 5 yr post-LVRS was noted in 73%, 46%, 35%, 27%, and 8% of all patients; decrease in dyspnea grade ≥ 1 in 88%, 69%, 46%, 27%, and 15%; and elimination of initial oxygen dependence in 18 patients in 78%, 50%, 33%, 22%, and 0%, respectively. Expiratory airflow improved due to the increase in both lung elastic recoil and small airway intraluminal caliber. Five patients decreased FEV_1 141 ± 60 mL/yr and FVC 102 ± 189 mL/yr over 3.8 ± 1.2 yr post-LVRS, similar to their pre-LVRS rate of decline. In the 11 patients who survived 5 yr, at 0.5-1.0 yr post-LVRS peak increase in FEV_1 was 438 ± 366 mL, with a decline of 149 ± 157 mL the following year and 78 ± 59 mL/yr over 4.0-4.5 yr. Bilateral LVRS provided palliative clinical and physiological improvement in 9 of 26 patients at 3 yr, 7 at 4 yr, and 2 at 5 yr.

Multivariable Assessment of the 6-Min Walking Test in Patients with Chronic Obstructive Pulmonary Disease—van Stel HF, Bogaard JM, Rijssenbeek-Nouwens LH, Colland VT. Am J Respir Crit Care Med 2001 Jun;163(7):1567-1571.

Functional exercise tolerance in patients with chronic obstructive pulmonary disease (COPD) is often assessed by the 6-min walking test (6MWT). To assess if the use of multiple factors adds to walking distance in describing performance in the 6MWT, an exploratory factor analysis was performed on physiological measurements and dyspnea ratings recorded during testing. Eighty-three patients with mild to severe COPD performed repeated 6MWTs before inpatient pulmonary rehabilitation. Factor analysis on 15 variables yielded a stable four-factor structure explaining 78.4% of the total variance. Recorded heart rate variables contributed to factor 1 (heart rate pattern), walking distance, heart rate increase, and decrease contributed to factor 2 (endurance capacity), oxygen desaturation variables contributed to factor 3 (impairment of oxygen transport), and dyspnea and effort variables contributed to factor 4 (perceived symptoms). Walking distance decreased in half of the 53 patients measured posttreatment, but self-perceived change in exercise tolerance improved in 84% and was explained by change in walking distance, by less desaturation, and by less dyspnea ($R^2 = 0.55$, $p = 0.005$). Qualitative analysis showed that 29 of 53 patients improved in three or four factors. Performance in the 6MWT can be described with four statistically independent and clinically interpretable factors. Because clinically relevant changes consist of more than only walking distance, assessment of functional exercise tolerance in patients with COPD improves by reporting multiple variables.

Influence of Tidal Volume on Alveolar Recruitment: Respective Role of PEEP and a Recruitment Maneuver—Richard JC, Maggiore SM, Jonson B, Mancebo J, Lemaire F, Brochard L. Am J Respir Crit Care Med 2001 Jun;163(7):1609-1613.

Both reduction in tidal volume (V_T) and alveolar recruitment may be important to limit ventilator-associated lung injury during mechanical ventilation of patients with the acute respiratory distress syndrome (ARDS). The aim of this study was to assess the risk of alveolar derecruitment associated with V_T reduction from 10 to 6 mL/kg. Whether this V_T -related derecruitment could be reversed, either by a recruitment maneuver or by an increase in positive end-expiratory pressure (PEEP) level, was also investigated. Fifteen patients with ARDS were successively ventilated using conventional V_T (CV_T = 10 ± 1 mL/kg) and low V_T (LV_T = 6 ± 1 mL/kg); total PEEP ($PEEP_{tot}$) was individually set at the lower inflection point (P_{lip}) of the pressure-volume curve ($PEEP_{tot}$ = 11 ± 4 cm H_2O). Pressure-volume curves were recorded from zero PEEP (ZEEP) and from PEEP, and recruited volume (V_{rec}) was calculated as the volume difference between the two curves for a given pressure. Despite a similar $PEEP_{tot}$, V_{rec} was significantly lower with LV_T than with CV_T, indicating low V_T -induced alveolar derecruitment. Reduction in V_T was associated with a reduced S_{aO_2}). In 10 patients, V_{rec} was also mea-

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sured before and after a recruitment maneuver (two sustained inflations at 45 cm H_2O), and after an increase in PEEP (by 4 cm H_2O). Low V_T-induced derecruitment was reversed by a recruitment maneuver and by increasing PEEP. We conclude that a reduction in V_T could be responsible for alveolar derecruitment, which may be transiently reversed by a reexpansion maneuver or prevented by a PEEP increase above P_{lip}.

Physiologic Effects of Negative Pressure Ventilation in Acute Exacerbation of Chronic Obstructive Pulmonary Disease—Gorini M, Corrado A, Villella G, Ginanni R, Augustynen A, Tozzi D. Am J Respir Crit Care Med 2001 Jun;163(7):1614-1618.

To assess the physiologic effects of continuous negative extrathoracic pressure (CNEP), negative pressure ventilation (NPV), and negative extrathoracic end-expiratory pressure (NEEP) added to NPV in patients with acute exacerbation of chronic obstructive pulmonary disease (COPD), we measured in seven patients ventilatory pattern, arterial blood gases, respiratory mechanics, and pressure- time product of the diaphragm (PTPdi) under four conditions: (1) spontaneous breathing (SB); (2) CNEP (-5 cm H_2O); (3) NPV; (4) NPV plus NEEP. CNEP and NPV were provided by a microprocessor-based iron lung capable of thermistor-triggering. Compared with SB, CNEP improved slightly but significantly P_{aCO_2} and pH, and decreased PTPdi (388 ± 59 versus 302 ± 43 cm H_2O·s, respectively, $p < 0.05$) and dynamic intrinsic positive end-expiratory pressure (PEEPi) (4.6 ± 0.5 versus 2.1 ± 0.3 cm H_2O, respectively, $p < 0.001$). NPV increased minute ventilation ($\dot{V}_E$), improved arterial blood gases, and decreased PTPdi to 34% of value during SB ($p < 0.001$). NEEP added to NPV further slightly decreased PTPdi and improved patient-ventilator interaction by reducing dynamic PEEPi and nontriggering inspiratory efforts. We conclude that CNEP and NPV, provided by microprocessor-based iron lung, are able to improve ventilatory pattern and arterial blood gases, and to unload inspiratory muscles in patients with acute exacerbation of COPD.

The Critical Nursing Situation Index for Safety Assessment in Intensive Care—Binnekade JM, de Mol BA, Kesecioglu J, de Haan RJ. Intensive Care Med 2001 Jun;27(6):1022-1028.

OBJECTIVE: The assessment of critical nursing situations can be a valuable tool in the detection of weak elements in the safety of patients and the quality of care in the ICU. A critical nursing situation can be defined as any observable situation, which deviates from good clinical practice and which may potentially lead to an adverse event. The aim of our study was to establish the feasibility, reliability and validity of the Critical Nursing Situation Index (CNSI) as a tool for assessing the safety and the quality of nursing in the ICU. DESIGN: We described the deviations from standards and protocols in daily ICU nursing care, selected those with an implicit, clear risk for the patients and translated them into explicitly observable items. If an item was applicable during observation of the ICU practice, a critical nursing situation could be recorded as either true or false. The reliability of the CNSI was defined in terms of inter-observer agreement. The validity was assessed by exploring the relationship between the nursing time available (more or less than 30 min per patient per hour) and the incidence of critical nursing situations. SETTING: The study was performed in the ICU of a teaching hospital (30 IC beds) in which all disciplines, including cardiothoracic surgery and neurosurgery, were represented. PATIENTS: The CNSI was randomly applied to 83 ICU patients over a period of 3 months (200 times). MEASUREMENTS AND RESULTS: The reliability of the index was substantial (Kappa values in the range ≥ 0.70 to > 0.80). In terms of validity, less nursing time resulted in more critical situations (pooled relative risk (RR) 1.36; 95% confidence limits 1.11/1.67). CONCLUSION: The CNSI is simple to use and has encouraging metric properties, whereas the assessments are closely related to direct patient care. Moreover, the CNSI provides a tool for safety assessment by monitoring potentially dangerous situations that are generally regarded as needing to be avoided.

Is a Short Trial of Prone Positioning Sufficient to Predict the Improvement in Oxygenation in Patients with Acute Respiratory Distress Syndrome?—Papazian L, Paladini MH, Bregeon F, Huiart L, Thirion X, Saux P, et al. Intensive Care Med 2001 Jun;27(6):1044-1049.

OBJECTIVE: To determine whether a 1-h trial of prone positioning is sufficient to identify responders. DESIGN: Prospective clinical cohort study in a medico-surgical ICU in a teaching hospital. PATIENTS: 49 patients with acute respiratory distress syndrome. INTERVENTIONS: A 6-h period of prone positioning. MEASUREMENTS AND RESULTS: Baseline measurements (blood gas analysis and respiratory parameters) were evaluated in supine position just prior to turning the patients prone. Measurements were then repeated 1 h after the beginning of prone positioning (PP1h) and at the end of the 6-h period of prone positioning (PP6h). The last measurements were performed 1 h after repositioning the patients supine. Prone position induced an increase in the P_{aO_2}/F_{IO_2} ratio ($p < 0.001$). A response (increase in P_{aO_2}/F_{IO_2} ratio of at least 20 % at PP1h and/or at PP6h) was observed in 37 of 49 patients (76%). Twenty-seven of these patients (73%) were responders at PP1h while 10 (27%) were responders only at PP6h. In all, two-thirds of the patients were considered persistent responders. However, whereas the P_{aO_2}/F_{IO_2} ratio decreased significantly 1 h after repositioning the fast responders supine, the P_{aO_2}/F_{IO_2} ratio remained unchanged after repositioning slow responders. CONCLUSIONS: A short-term trial of prone positioning does not appear a sufficient method to identify patients who would benefit from the postural treatment.

Efficacy of Early Piston-Type High-Frequency Oscillatory Ventilation in Infants with Respiratory Distress Syndrome—Sakai T, Kamohara T, Aiba S, Yoshioka T, Itinohe A, Chiba H, et al. Pediatr Pulmonol 2001 Aug;32(2):168-174.

We investigated whether the combination of surfactant replacement therapy and early application of high-frequency oscillatory ventilation (HFOV) was more effective in patients with respiratory distress syndrome (RDS) than late application of HFOV and conventional mechanical ventilation (CMV). To determine this, we retrospectively reviewed the cases of 126 neonates with RDS who received surfactant replacement therapy within 4 hr after birth. Patients were grouped into those who received HFOV immediately after birth (HFOV group), those who initially were ventilated by CMV and subsequently received HFOV (CMV/HFOV group), and those who did not receive HFOV (CMV group). Changes in respiratory system compliance (Crs), arterial-alveolar oxygen gradient (a/ApO_2), and mean airway pressure (MAP) were compared. Infants who received HFOV were less mature than those who received CMV. The a/ApO_2 measured immediately after birth before surfactant replacement therapy was significantly lower in the HFOV and CMV/HFOV group than in the CMV group. After 72 hr, the Crs in the HFOV group was higher than in any other group and was significantly higher than the CMV/HFOV group at 48 and 120 hr. These results suggest that initiating HFOV in combination with surfactant replacement therapy immediately after birth provides effective ventilatory support for infants with RDS.

Childhood Outcome After Early High-Frequency Oscillatory Ventilation for Neonatal Respiratory Distress Syndrome—Gerstmann DR, Wood K, Miller A, Steffen M, Ogden B, Stoddard RA, Minton SD. Pediatrics 2001 Sep;108(3):617-623.

OBJECTIVE: In a previous multicenter controlled clinical trial, we randomly assigned surfactant treated premature newborns with moderate to severe respiratory distress syndrome to early treatment with high-frequency oscillatory ventilation (HFOV) or to conventional ventilation (CV). Compared with control infants who were treated with CV, neonates who were treated with HFOV using a strategy designed to recruit and maintain lung volume and minimize oxygen exposure had clinical evidence of improved pulmonary outcome and less lung injury. We report a follow-up study designed to deter-

mine whether clinical differences persisted between these study groups. METHODS: Patients were recruited from 81 survivors at 1 center (Provo, Utah) and evaluated for sociodemographic and health history, growth, mental development, motor proficiency, and pulmonary function. RESULTS: Eighty-seven percent of the cohort who originally were assigned to treatment with HFOV (n = 36) or CV (n = 33) were seen in follow-up at a mean age of 77 months (6.4 years). There were no differences in the frequency of hospitalization, pulmonary illness, asthma, or disabilities. Growth, verbal IQ, and motor development were appropriate for age and not different between groups. Patients who initially were randomized to treatment with CV showed pulmonary function evidence of decreased peak expiratory flow, increased residual lung volume, and maldistribution of ventilation. CONCLUSION: Neurodevelopmental childhood outcome after early intervention HFOV was normal and not different compared with patients who were treated with CV. Surfactant replacement combined with early HFOV using a lung recruitment strategy ameliorates the acute lung injury in respiratory distress syndrome that predisposes some preterm infants to develop chronic lung disease.

End-of-Life Care for Neonates and Infants: The Experience and Effects of a Palliative Care Consultation Service—Pierucci RL, Kirby RS, Leuthner SR. Pediatrics 2001 Sep;108(3):653-660.

OBJECTIVE: Neonates and infants have the highest death rate in the pediatric population, yet there is a paucity of data about their end-of-life care and whether a palliative care service can have an impact on that care. The objective of this study was to describe end-of-life care for infants, including analysis of palliative care consultations conducted in this population. We hypothesized that the palliative care consultations performed had an impact on the infants' end-of-life care. DESIGN: A retrospective chart review using the "End of Life Chart Review" from the Center to Improve Care for the Dying was conducted. The participants were the patients at Children's Hospital of Wisconsin who died at <1 year of age during the 4-year period between January 1, 1994, and December 31, 1997. The patients' place of death, medical interventions performed, and emotionally supportive services provided to families were analyzed. RESULTS: Among the 196 deaths during the study period, 25 (13%) of these infants and families had palliative care consultations. The rate of consultations increased from 5% of the infant deaths in 1994 to 38% of the infant deaths in 1997. Infants of families that received consultations had fewer days in intensive care units, blood draws, central lines, feeding tubes, vasopressor and paralytic drug use, mechanical ventilation, cardiopulmonary resuscitation, and x-rays, and the families had more frequent referrals for chaplains and social services than families that did not have palliative care consultations. CONCLUSIONS: This study describes the end-of-life care that infants and their families received. Fewer medical procedures were performed, and more supportive services were provided to infants and families that had a palliative care consultation. This suggests that palliative care consultation may enhance end-of-life care for newborns.

Work of Breathing During Constant- and Variable-Flow Nasal Continuous Positive Airway Pressure in Preterm Neonates—Pandit PB, Courtney SE, Pyon KH, Saslow JG, Habib RH. Pediatrics 2001 Sep;108(3):682-685.

BACKGROUND: Constant-flow nasal continuous positive airway pressure (NCPAP) often is used in preterm neonates to recruit and maintain lung volume. Physical model studies indicate that a variable-flow NCPAP device provides more stable volume recruitment with less imposed work of breathing (WOB). Although superior lung recruitment with variable-flow NCPAP has been demonstrated in preterm neonates, corroborating WOB data are lacking. OBJECTIVE: To measure and compare WOB associated with the use of variable-flow versus constant-flow NCPAP in preterm neonates. METHODS: Twenty-four preterm infants who were receiving constant-flow NCPAP (means, SD) and had birth weight of 1024 ± 253 g, gestational age of 28 ± 1.7 weeks, age of 14 ± 13 days, and F_{IO_2} of 0.3 ± 0.1 were studied. Variable-flow and constant-flow NCPAP were applied in random order. We measured changes in lung volume and tidal ventilation (V_T) by DC-coupled/calibrated respiratory inductance plethysmography as well as esophageal pressures at NCPAP of 8, 6, 4, and 0 cm H_2O. Inspiratory WOB (WOB_I) and lung compliance were calculated from the esophageal pressure and V_T data using standard methods. WOB was divided by V_T to standardize the results. RESULTS: WOB_I decreased at all CPAP levels with variable-flow NCPAP, with a maximal decrease at 4 cm H_2O. WOB_I increased at all CPAP levels with constant-flow CPAP. Lung compliance increased at all NCPAP levels with variable-flow, with a relative decrease at 8 cm H_2O, whereas it increased only at 8 cm H_2O with constant-flow NCPAP. Compared with constant-flow NCPAP, WOB_I was 13% to 29% lower with variable-flow NCPAP. CONCLUSION: WOB_I is decreased with variable-flow NCPAP compared with constant-flow NCPAP. The increase in WOB_I with constant-flow NCPAP indicates the presence of appreciable imposed WOB with this device. Our study, performed in neonates with little lung disease, indicates the possibility of lung overdistention at CPAP of 6 to 8 cm H_2O with the variable-flow device. Further study is necessary to determine the efficacy of variable-flow NCPAP in neonates with significant lung disease and its use over extended periods of time.

Effects of PEEP on Inspiratory Resistance in Mechanically Ventilated COPD-Patients—Guerin C, Fournier G, Milic-Emili J. Eur Respir J 2001 Sep;18(3):491-498.

This study aimed to investigate the effect of increased lung volume with positive end-expiratory pressure (PEEP) on respiratory resistance in patients

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with chronic obstructive pulmonary disease (COPD). Ten patients with COPD were mechanically ventilated for acute respiratory failure. PEEP was set at 0, 5, 10 and 15 cm H_2O. Using the rapid airway occlusion technique, the total inspiratory resistance of the respiratory system was partitioned into interrupter (Rint,rs) and additional effective (ΔRrs) resistances. At each level of PEEP, at constant inflation flow, the inflation volume ($\Delta\dot{V}$) was varied from 0.2-1 L, and, at constant $\Delta\dot{V}$, the inflation flow was varied from 0.2-1.2 L x s^{-1}. The changes in end-expiratory lung volume (ΔEELV̇) induced by PEEP were also measured. The difference between the EELV̇ and the relaxation volume of the respiratory system (ΔFRC) increased significantly with PEEP of 10 and 15 cm H_2O as compared to a PEEP of 0, the increase being associated with a significant reduction of Rint,rs. By contrast, ΔRrs was independent of ΔFRC. At constant $\Delta\dot{V}$, Rint,rs fitted Rohrer's equation (Rint,rs = K1 + K2 x flow). While K2 significantly declined with ΔFRC, K1 did not change. At all levels of PEEP, ΔRrs was not influenced by ΔFRC. With increasing lung volume induced by positive end-expiratory pressure, the inspiratory airway resistance decreased, whereas the viscoelastic behaviour of the respiratory system, as reflected by additional effective resistance, did not change.

Effectiveness of CPAP Treatment in Daytime Function in Sleep Apnea Syndrome: A Randomized Controlled Study with an Optimized Placebo—Montserrat JM, Ferrer M, Hernandez L, Farre R, Vilagut G, Navajas D, et al. Am J Respir Crit Care Med 2001 Aug 15;164(4):608-613.

Application of continuous positive airway pressure (CPAP) as the standard treatment for sleep apnea/hypopnea syndrome (SAHS) is a moot point. Studies on the effectiveness of this treatment have been challenged because of the lack of a suitable placebo. The recent description of a true placebo (sham CPAP) prompted us to conduct a randomized trial of CPAP or placebo to assess the effectiveness of CPAP in improving SAHS-related symptoms and daytime function in patients with moderate to severe SAHS. Forty-eight patients, stratified in four groups according to severity, were randomly allocated into two treatment groups (optimal and sham CPAP) for a 6-wk period. Of these, 45 completed follow-up (91% males; age: 54 ± 10 yr; body mass index [BMI]: 32 ± 6 kg/m^2; apnea-hypopnea index [AHI]: 54 ± 19 events/h; and Epworth Sleepiness Scale [ESS]: 16 ± 5). The ESS, a questionnaire on SAHS-related symptoms, Functional Outcomes Sleep Questionnaire (FOSQ), and the Short Form Health Survey (SF-36) were completed at inclusion and after treatment. After 10 d of washout, the placebo group was treated with optimal CPAP and reassessed before and after optimal CPAP. The group receiving optimal CPAP when compared with the group with sham CPAP showed considerably greater improvement in the relief of sleepiness (-9.5 versus -2.3, $p < 0.001$), other SAHS-related symptoms (-18.5 versus -4.5, $p < 0.001$), vigilance (+8.5 versus +3.4, $p = 0.009$), and general productivity (+4.0 versus +0.5, $p = 0.04$) FOSQ scales. Both groups used a similar number of hours for the optimal and the sham CPAP (4.3 versus 4.5, (p = NS). The patients initially treated with placebo CPAP improved significantly more when optimal CPAP was applied for ESS (-2.3 versus -6.7, $p < 0.001$) and other sleep apnea syndrome (SAS)-related symptoms (-4.5 versus -11.2, $p = 0.02$). Our study provides strong evidence of the effectiveness of CPAP treatment in improving symptoms and perceived health status in moderate to severe SAHS.

Adaptive Pressure Support Servo-Ventilation: A Novel Treatment for Cheyne-Stokes Respiration in Heart Failure—Teschler H, Dohring J, Wang YM, Berthon-Jones M. Am J Respir Crit Care Med 2001 Aug 15;164(4):614-619.

Adaptive servo-ventilation (ASV) is a novel method of ventilatory support designed for Cheyne-Stokes respiration (CSR) in heart failure. The aim of our study was to compare the effect of one night of ASV on sleep and breathing with the effect of other treatments. Fourteen subjects with stable cardiac failure and receiving optimal medical treatment were tested untreated and on four treatment nights in random order: nasal oxygen (2 L/min), continuous positive airway pressure (CPAP) (mean 9.25 cm H_2O), bilevel (mean 13.5/5.2 cm H_2O), or ASV largely at the default settings (mean pressure 7 to 9 cm H_2O) during polysomnography. Thermistor apnea + hypopnea index (AHI) declined from 44.5 ± 3.4/h (SEM) untreated to 28.2 ± 3.4/h oxygen and 26.8 ± 4.6/h CPAP (both $p < 0.001$ versus control), 14.8 ± 2.3/h bilevel, and 6.3 ± 0.9/h ASV ($p < 0.001$ versus bilevel). Effort band AHI behaved similarly. Arousal index decreased from 65.1 ± 3.9/h untreated to 29.8 ± 2.8/h oxygen and 29.9 ± 3.2/h CPAP, to 16.0 ± 1.3/h bilevel and 14.7 ± 1.8/h ASV ($p < 0.01$ versus all except bilevel). There were large increases in slow-wave and rapid eye movement (REM) sleep with ASV but not with oxygen or CPAP. All subjects preferred ASV to CPAP. One night ASV suppresses central sleep apnea and/or CSR (CSA/CSR) in heart failure and improves sleep quality better than CPAP or 2 L/min oxygen.

Helium-Oxygen in the Postextubation Period Decreases Inspiratory Effort—Jaber S, Carlucci A, Boussarsar M, Fodil R, Pigeot J, Maggiore S, et al. Am J Respir Crit Care Med 2001 Aug 15;164(4):633-637.

After tracheal extubation, upper and total airway resistances may frequently be increased resulting in an increase in inspiratory effort to breathe. We tested whether breathing a helium-oxygen mixture (HeO_2) would reduce inspiratory effort in the period after extubation. Eighteen consecutive patients with no chronic obstructive pulmonary disease who had received mechanical ventilation (> 48 h) were successively studied immediately after extubation (N_2O_2), 15 min after breathing HeO_2, and after return to N_2O_2. Effort to breathe, assessed by the transdiaphragmatic pressure swings (ΔPdi) and the pressure-time index of the diaphragm (PTI), comfort, and gas exchange, were the main end points. The mean reduction of the transdiaphragmatic pressure under HeO_2 was 19 ± 5%. All but three patients presented a decrease in transdiaphragmatic pressure under HeO_2, ranging from - 4 to - 55%, and a significant reduction in ΔPdi was observed between HeO_2 and N_2O_2 (10.2 ± 0.7 versus 8.6 ± 1.1 versus 10.0 ± 0.8 cm H_2O for the three consecutive periods; $p < 0.05$). PTI also differed significantly between HeO_2 and N_2O_2 (197 ± 19 versus 166 ± 22 versus 201 ± 23 cm H_2O/s/min for the three periods; $p < 0.05$). Breathing HeO_2 significantly improved comfort, whereas gas exchange was not modified. We conclude that the use of HeO_2 in the immediate postextubation period decreases inspiratory effort and improves comfort.

Physiological Response to Pressure Support Ventilation Delivered Before and After Extubation in Patients Not Capable of Totally Spontaneous Autonomous Breathing—Vitacca M, Ambrosino N, Clini E, Porta R, Rampulla C, Lanini B, Nava S. Am J Respir Crit Care Med 2001 Aug 15; 164(4):638-641.

We designed a prospective, physiological study in 12 patients affected by chronic respiratory disorders. The study was aimed at assessing the diaphragm energy expenditure (PTPdi), lung resistance (RL) and elastance (EL), arterial blood gases (ABG), breathing pattern, and dyspnea measured by a visual analog scale during invasive pressure support ventilation (i-PSV) and noninvasive PSV (n-PSV). The ventilator settings were kept the same. Both i-PSV and n-PSV significantly reduced the PTPdi per minute, compared with that during a T-piece trial (204.4 ± 93.8 cm H_2O x s/min [i-PSV]; 197.5 ± 119.8 [n-PSV]; 393.8 ± 129.0 [T-piece]). Expired tidal volume (V_{Te}) was significantly higher ($p < 0.05$) during n-PSV (615 ± 166 mL) than during i-PSV (519 ± 140 mL). The respiratory pump (PTPdi/V_{Te}) was more effective ($p < 0.05$) with noninvasive ventilation (22.3 ± 2.3 cm H_2O x s/L for i-PSV versus 17.2 ± 3.3 for n-PSV). RL and EL were similar with the two modes of ventilation. Overall dyspnea was significantly ($p < 0.05$) better during n-PSV than i-PSV, whereas ABG were similar. We have shown, in patients affected by stable chronic respiratory disorders not ready to sustain totally spontaneous breathing, that i-PSV and n-PSV are equally effective in reducing the PTPdi and in improving ABG, but that n-PSV seems to be better tolerated.

Virtual Tools for Imaging of the Thorax—Ferretti GR, Bricault I, Coulomb M. Eur Respir J 2001 Aug;18(2):381-392.

Helical computed tomography (HCT) allows for volume acquisition of the entire thorax during a single apnoea. Combination of HCT acquisition with synchronous vascular enhancement gives rise to HCT angiography (HCTA). In the last decade, HCT and HCTA have revolutionized the diagnosis of thoracic diseases, modifying many diagnostic algorithms. Because HCT provides for a true volume acquisition free of respiratory misregistration, three-dimensional (3D) rendering techniques can be applied to HCT acquisitions. As these 3D rendering techniques present the HCT information in a different format to the conventional transaxial CT slices, they can be summarized as virtual tools. The purpose of this review is to give the readers the most important technical aspects of virtual tools, to report their application to the thorax, to answer clinical and scientific questions, and to stress their importance for patient management, clinical decision making, and research.

Comparison of Two Methods for Weaning Patients with Chronic Obstructive Pulmonary Disease Requiring Mechanical Ventilation for More Than 15 days—Vitacca M, Vianello A, Colombo D, Clini E, Porta R, Bianchi L, et al. Am J Respir Crit Care Med 2001 Jul 15;164(2):225-230.

We designed a prospective multicenter randomized controlled study in three long-term weaning units (LWU) to evaluate which protocol, inspiratory pressure support ventilation (PSV) or spontaneous breathing trials (SB), is more effective in weaning patients with chronic obstructive pulmonary disease (COPD) requiring mechanical ventilation for more than 15 d. Fifty-two of 75 patients, failing an initial T-piece trial at admission, were randomly assigned to PSV or SB (26 in both groups). No significant difference was found in weaning success rate (73% versus 77% in the PSV and SB group, respectively), mortality rate (11.5% versus 7.6%), duration of ventilatory assistance (181 ± 161 versus 130 ± 106 h), LWU (33 ± 12 versus 35 ± 19 d), or total hospital stay. The results of these defined protocols were retrospectively compared with an "uncontrolled clinical practice" in weaning historical control patients. The overall 30-d weaning success rate was significantly greater (87% versus 70%) and the time spent under mechanical ventilation by survived and weaned patients was shorter in the patients in the study than in historical control patients (103 ± 144 versus 170 ± 127 h). The LWU and hospital stays were also significantly shorter (27 ± 12 versus 38 ± 18 and 38 ± 17 versus 47 ± 18 d). Spontaneous breathing trials and decreasing levels of PSV are equally effective in difficult-to-wean patients with COPD. The application of a well-defined protocol, independent of the mode used, may result in better outcomes than uncontrolled clinical practice.

Efficacy of Low Tidal Volume Ventilation in Patients with Different Clinical Risk Factors for Acute Lung Injury and the Acute Respiratory Distress Syndrome—Eisner MD, Thompson T, Hudson LD, Luce JM, Hayden D, Schoenfeld D, Matthay MA. Am J Respir Crit Care Med 2001 Jul 15;164(2):231-236.

In patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS), a recent ARDS Network randomized controlled trial demonstrated that a low tidal volume (V_T) mechanical ventilation strategy (6 mL/kg) reduced mortality by 22% compared with traditional mechanical ventilation (12 mL/kg). In this study, we examined the relative efficacy of low V_T mechanical ventilation among 902 patients with different clinical risk factors for ALI/ARDS who participated in ARDS Network randomized controlled trials. The clinical risk factor for ALI/ARDS was associated with substantial variation in mortality. The risk of death (before discharge home with unassisted breathing) was highest in patients with sepsis (43%); intermediate in subjects with pneumonia (36%), aspiration (37%), and other risk factors (35%); and lowest in those with trauma (11%) ($p < 0.0001$). Despite these differences in mortality, there was no evidence that the efficacy of the low V_T strategy varied by clinical risk factor ($p = 0.76$, for interaction between ventilator group and risk factor). There was also no evidence of differential efficacy of low V_T ventilation in the other study outcomes: proportion of patients achieving unassisted breathing ($p = 0.59$), ventilator-free days ($p = 0.58$), or development of nonpulmonary organ failure ($p = 0.44$). Controlling for demographic and clinical covariates did not appreciably affect these results. After reclassifying the clinical risk factors as pulmonary versus nonpulmonary predisposing conditions and infection-related versus non-infection-related conditions, there was still no evidence that the efficacy of low V_T ventilation differed among clinical risk factor subgroups. In conclusion, we found no evidence that the efficacy of the low V_T ventilation strategy differed among clinical risk factor subgroups for ALI/ARDS.

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Short-Term Effects of Inhaled Nitric Oxide and Prone Position in Pulmonary and Extrapulmonary Acute Respiratory Distress Syndrome—Rialp G, Betbese AJ, Perez-Marquez M, Mancebo J. Am J Respir Crit Care Med 2001 Jul 15;164(2):243-249.

Inhaled nitric oxide (NO) and prone position (PP) are frequently used in the treatment of acute respiratory distress syndrome (ARDS). We compared the gas exchange and hemodynamic effects induced by the combination of NO inhalation and PP in patients with ARDS and analyzed whether or not pulmonary (Pu) and extrapulmonary (Epu) ARDS patients behave differently. Eight Pu and seven Epu ARDS patients were studied in four situations: supine position (SP); SP with NO inhalation at 5 ppm (SP + NO); PP; and PP with NO inhalation (PP + NO). In comparison with SP, NO inhalation and PP induced significant increases in P_{aO_2}/F_{IO_2} (from 106 ± 58 in SP to 131 ± 69 mm Hg in SP + NO, $p = 0.01$, and to 184 ± 67 mm Hg in PP, $p < 0.001$).

Pu and Epu ARDS showed a similar improvement in P_{aO_2}/F_{IO_2} with PP. Only Pu ARDS patients showed a significant increase ($p < 0.001$) in oxygenation induced by NO inhalation from 81 ± 45 to 100 ± 50 mm Hg in SP, and from 146 ± 53 to 197 ± 98 mm Hg in PP. In conclusion, PP is associated with a marked improvement in oxygenation, irrespective of the causes of ARDS, and additive effects of NO inhalation are mainly seen in patients with Pu ARDS.

Gel Lubrication of the Tracheal Tube Cuff Reduces Pulmonary Aspiration—Blunt MC, Young PJ, Patil A, Haddock A. Anesthesiology 2001 Aug;95(2):377-381.

BACKGROUND: Leakage of fluid occurs along the longitudinal folds within the wall of an inflated high-volume, low-pressure cuff. Theoretically, lubrication of the cuff with a water-soluble gel might prevent aspiration by plugging the channels in the cuff wall. Pulmonary aspiration during anesthesia has been linked with postoperative pneumonia and during critical illness causes ventilator-associated pneumonia. METHODS: Lubricated cuffs were compared with nonlubricated cuffs for leakage of dye placed in the subglottic space to the tracheobronchial tree in a benchtop model (n = 5) and in a prospective double-blinded randomized controlled trial of anesthetized patients (n = 36). The duration of the efficacy of the lubricant was determined in a prospective open observational study of critically ill patients with tracheostomies (n = 9). Dye was detected clinically by dye coloration of secretions during tracheal suctioning. RESULTS: In the benchtop model the incidence of leakage was 0% in the lubrication group and 100% in the nonlubrication group ($p < 0.01$). Dye leakage in anesthetized patients was 11% in the lubrication group and 83% in the nonlubrication group ($p < 0.0001$). In the critically ill patients with lubricated cuffed tracheostomy tubes, leakage first occurred after a median period of 48 h (range, 24-120 h). CONCLUSIONS: Cuff lubrication with a water-soluble gel reduces pulmonary aspiration in anesthetized patients. In the critically ill patient with a tracheostomy the protective effect is lost after 24-120 h.

Cardiorespiratory Effects of Automatic Tube Compensation During Airway Pressure Release Ventilation in Patients with Acute Lung Injury—Wrigge H, Zinserling J, Hering R, Schwalfenberg N, Stuber F, von Spiegel T, et al. Anesthesiology 2001 Aug;95(2):382-389.

BACKGROUND: Spontaneous breaths during airway pressure release ventilation (APRV) have to overcome the resistance of the artificial airway. Automatic tube compensation provides ventilatory assistance by increasing airway pressure during inspiration and lowering airway pressure during expiration, thereby compensating for resistance of the artificial airway. The authors studied if APRV with automatic tube compensation reduces the inspiratory effort without compromising cardiovascular function, end-expiratory lung volume, and gas exchange in patients with acute lung injury. METHODS: Fourteen patients with acute lung injury were breathing spontaneously during APRV with or without automatic tube compensation in random order. Airway pressure, esophageal and abdominal pressure, and gas flow were continuously measured, and tracheal pressure was estimated. Transdiaphragmatic pressure time product was calculated. End-expiratory lung volume was determined by nitrogen washout. The validity of the tracheal pressure calculation was investigated in seven healthy ventilated pigs. RESULTS: Automatic tube compensation during APRV increased airway pressure amplitude from 7.7±1.9 to 11.3±3.1 cm H_2O (mean ± SD; $p < 0.05$) while decreasing transdiaphragmatic pressure time product from 45±27 to 27±15 cm $H_2O \times s^{-1} \times min^{-1}$ ($p < 0.05$), whereas tracheal pressure amplitude remained essentially unchanged (10.3±3.5 vs. 10.1±3.5 cm H_2O). Minute ventilation increased from 10.4±1.6 to 11.4±1.5 L/min ($p < 0.001$), decreasing arterial carbon dioxide tension from 52±9 to 47±6 mmHg ($p < 0.05$) without affecting arterial blood oxygenation or cardiovascular function. End-expiratory lung volume increased from 2,806±991 to 3,009±994 mL ($p < 0.05$). Analysis of tracheal pressure-time curves indicated nonideal regulation of the dynamic pressure support during automatic tube compensation as provided by a standard ventilator. CONCLUSION: In the studied patients with acute lung injury, automatic tube compensation markedly unloaded the inspiratory muscles and increased alveolar ventilation without compromising cardiorespiratory function and end-expiratory lung volume.

Preserved Spontaneous Breathing Improves Cardiac Output During Partial Liquid Ventilation—Franz AR, Mack C, Reichart J, Pohlandt F, Hummler HD. Am J Respir Crit Care Med 2001 Jul 1;164(1):36-42.

The aim of this study was to examine whether preserved spontaneous breathing (SB) supported by proportional-assist ventilation (PAV) would improve cardiac output (CO) during partial liquid ventilation (PLV) in rabbits with and without lung disease if compared with time-cycled, volume-controlled ventilation (CV) combined with muscle paralysis (MP). PLV was initiated in 17 healthy rabbits and 17 surfactant-depleted rabbits using 12 to 15 mL/kg of perfluorodecaline. Both ventilatory modes, SB+PAV and CV+MP, were applied in random sequence using a crossover design. CO was measured by thermodilution. CO was significantly higher during SB+PAV than during CV+MP: 136 ± 21 mL/kg x min (mean ± SD) versus 120 ± 30 mL/kg x min ($p = 0.004$) in healthy rabbits, and 147 ± 19 mL/kg x min versus 111 ± 13 mL/kg x min ($p < 0.0001$) in surfactant-depleted rabbits, resulting in an improved oxygen delivery. This difference was mainly caused by a larger stroke volume during SB+PAV, whereas there was little change in heart rate. In surfactant-depleted rabbits, SB+PAV resulted in improved arterial blood pressure and arterial and mixed venous pH and in a higher P_{aO_2} at the same level of PEEP and mean airway pressure. We conclude that during PLV, CO is higher during SB+PAV than during CV+MP, resulting in an improved oxygen delivery. In surfactant-depleted rabbits, improved CO, oxygen delivery, and arterial blood pressure resulted in higher pH, possibly reflecting improved tissue perfusion and oxygenation.

Prevalence of Upper Respiratory Symptoms in Patients with Symptomatic Gastroesophageal Reflux Disease—Theodoropoulos DS, Ledford DK, Lockey RF, Pecoraro DL, Rodriguez JA, Johnson MC, Boyce HW Jr. Am J Respir Crit Care Med 2001 Jul 1;164(1):72-76.

This study evaluated the prevalence of upper respiratory symptoms (URS) among patients with symptomatic gastroesophageal reflux disease (GERD). Seventy-four subjects with heartburn completed a URS questionnaire before dual-probe, 24-h esophageal pH monitoring. The URS questionnaire was also completed by 74 normal volunteers without previous or current symptoms of GERD. Esophageal pH monitoring results were classified as normal, distal, or proximal and distal gastroesophageal reflux using standardized criteria. Mean URS scores (± SD) were 8.31 ± 3.98 in the 52 subjects with GERD and 4.57 ± 3.57 in the 22 subjects with negative pH probe studies, $p = 0.02$. Subjects with negative pH probe studies and normal volunteers scored similarly on the URS questionnaire. Reflux episodes/24 h correlated with URS scores, $r = 0.47$, $p = 0.0001$. Seventy-five percent of subjects with upper reflux, 68% of subjects with lower reflux, 36% of subjects with normal esophageal pH studies, and 9% of normal volunteers reported laryngeal symptoms for at least 5 d/mo. Sixty-nine percent of subjects with upper reflux, 50% of subjects with lower reflux, 31% of subjects with normal pH studies, and 14% of normal volunteers reported nasal symptoms for at least 5 d/mo. URS are frequent among subjects with GERD.

The Influence of Hospital Volume on Survival After Resection for Lung Cancer—Bach PB, Cramer LD, Schrag D, Downey RJ, Gelfand SE, Begg CB. N Engl J Med 2001 Jul 19;345(3):181-188.

BACKGROUND: Among patients who have undergone high-risk operations for cancer, postoperative mortality rates are often lower at hospitals where more of these procedures are performed. We undertook a population-based study to estimate the extent to which the number of procedures per-

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formed at a hospital (hospital volume) is associated with survival after resection for lung cancer. METHODS: We studied patients 65 years old or older who received a diagnosis of stage I, II, or IIIA non-small-cell lung cancer between 1985 and 1996, resided in 1 of the 10 study areas covered by the Surveillance, Epidemiology, and End Results Program, and underwent surgery at a hospital that participates in the Nationwide Inpatient Sample (2118 patients and 76 hospitals). RESULTS: The volume of procedures at the hospital was positively associated with the survival of patients ($p < 0.001$). Five years after surgery, 44 percent of patients who underwent operations at the hospitals with the highest volume were alive, as compared with 33 percent of those who underwent operations at the hospitals with the lowest volume. Patients at the highest-volume hospitals also had lower rates of postoperative complications (20 percent vs. 44 percent) and lower 30-day mortality (3 percent vs. 6 percent) than those at the lowest-volume hospitals. CONCLUSIONS: Patients who undergo resection for lung cancer at hospitals that perform large numbers of such procedures are likely to survive longer than patients who have such surgery at hospitals with a low volume of lung-resection procedures.

Outcome of Adult Cardiopulmonary Resuscitations at a Tertiary Referral Center Including Results of "Limited" Resuscitations—Dumot JA, Burval DJ, Sprung J, Waters JH, Mraovic B, Karafa MT, et al. Arch Intern Med 2001 Jul 23;161(14):1751-1758.

BACKGROUND: The results of in-hospital resuscitations may depend on a variety of factors related to the patient, the environment, and the extent of resuscitation efforts. We studied these factors in a large tertiary referral hospital with a dedicated certified resuscitation team responding to all cardiac arrests. METHODS: Statistical analysis of 445 prospectively recorded resuscitation records of patients who experienced cardiac arrest and received advanced cardiac life support resuscitation. We also report the outcomes of an additional 37 patients who received limited resuscitation efforts because of advance directives prohibiting tracheal intubation, chest compressions, or both. MAIN OUTCOME MEASURES: Survival immediately after resuscitation, at 24 hours, at 48 hours, and until hospital discharge. RESULTS: Overall, 104 (23%) of 445 patients who received full advanced cardiac life support survived to hospital discharge. Survival was highest for patients with primary cardiac disease (30%), followed by those with infectious diseases (15%), with only 8% of patients with end-stage diseases surviving to hospital discharge. Neither sex nor age affected survival. Longer resuscitations, increased epinephrine and atropine administration, multiple defibrillations, and multiple arrhythmias were all associated with poor survival. Patients who experienced arrests on a nursing unit or intensive care unit had better survival rates than those in other hospital locations. Survival for witnessed arrests (25%) was significantly better than for nonwitnessed arrests (7%) ($p = 0.005$). There was a disproportionately high incidence of nonwitnessed arrests during the night (12 AM to 6 AM) in unmonitored beds, resulting in uniformly poor survival to hospital discharge (0%). None of the patients whose advance directives limited resuscitation survived. CONCLUSIONS: Very ill patients in unmonitored beds are at increased risk for a nonwitnessed cardiac arrest and poor resuscitation outcome during the night. Closer vigilance of these patients at night is warranted. The outcome of limited resuscitation efforts is very poor.

The Effect of BCG Vaccination on Tuberculin Reactivity and the Booster Effect Among Hospital Employees—Moreno S, Blazquez R, Novoa A, Carpena I, Menasalvas A, Ramirez C, Guerrero C. Arch Intern Med 2001 Jul 23;161(14):1760-1765.

BACKGROUND: We estimated the effect of remote BCG vaccination on tuberculin reactivity and the booster effect among hospital employees. METHODS: Cross-sectional survey at a university hospital. All personnel employed during a 24-month period were included in the study. Employees were administered 2-step tuberculin testing, and BCG vaccination scars were verified. RESULTS: Of 665 hospital employees studied, 239 (36%) had been vaccinated with BCG in childhood. Significant tuberculin reactions (≥ 5 mm) were more frequent among BCG-vaccinated (60%) than among nonvaccinated (29%) employees (odds ratio [OR], 3.6; 95% confidence interval [CI], 2.6-5.2). The predictive value of tuberculosis infection increased with increasing reaction size and greater age (from 37% in subjects 30 years or younger with indurations ≥ 5 mm to 100% in subjects 50 years or older with indurations ≥ 15 mm). Among 374 employees with a negative tuberculin test reaction who underwent a second test, 39 (43%) of 91 vaccinated subjects had a positive booster reaction in contrast to 51 (22%) of 232 nonvaccinated subjects (OR, 3.4; 95% CI, 2-5.7). Neither different size criteria nor different definitions of the booster effect had an impact on the predictive value of tuberculosis infection. CONCLUSIONS: Remote BCG vaccination largely influences the tuberculin reaction and the boosting phenomenon among hospital employees. The interpretation of the results of 2-step tuberculin testing in a BCG-vaccinated subject must take into account age, size of the reaction, and local prevalence of tuberculosis infection. No single criterion, however, can accurately separate reactions caused by true infection from those caused by BCG vaccination.

Excluding Pulmonary Embolism at the Bedside Without Diagnostic Imaging: Management of Patients with Suspected Pulmonary Embolism Presenting to the Emergency Department by Using a Simple Clinical Model and D-Dimer—Wells PS, Anderson DR, Rodger M, Stiell I, Dreyer JF, Barnes D, et al. Ann Intern Med 2001 Jul 17;135(2):98-107.

BACKGROUND: The limitations of the current diagnostic standard, ventilation-perfusion lung scanning, complicate the management of patients with suspected pulmonary embolism. We previously demonstrated that determining the pretest probability can assist with management and that the high negative predictive value of certain D-dimer assays may simplify the diagnostic process. OBJECTIVE: To determine the safety of using a simple clinical model combined with D-dimer assay to manage patients presenting to the emergency department with suspected pulmonary embolism. DESIGN: Prospective cohort study. SETTING: Emergency departments at four tertiary care hospitals in Canada. PATIENTS: 930 consecutive patients with suspected pulmonary embolism. INTERVENTIONS: Physicians first used a clinical model to determine patients' pretest probability of pulmonary embolism and then performed a D-dimer test. Patients with low pretest probability and a negative D-dimer result had no further tests and were considered to have a diagnosis of pulmonary embolism excluded. All other patients underwent ventilation-perfusion lung scanning. If the scan was nondiagnostic, bilateral deep venous ultrasonography was done. Whether further testing (by serial ultrasonography or angiography) was done depended on the patients' pretest probability and the lung scanning results. MEASUREMENTS: Patients received a diagnosis of pulmonary embolism if they had a high-probability ventilation-perfusion scan, an abnormal result on ultrasonography or pulmonary angiography, or a venous thromboembolic event during follow-up. Patients for whom the diagnosis was considered excluded were followed up for 3 months for the development of thromboembolic events. RESULTS: The pretest probability of pulmonary embolism was low, moderate, and high in 527, 339, and 64 patients (1.3%, 16.2%, and 37.5% had pulmonary embolism), respectively. Of 849 patients in whom a diagnosis of pulmonary-embolism had initially been excluded, 5 (0.6% [95% CI, 0.2% to 1.4%]) developed pulmonary embolism or deep venous thrombosis during follow-up. However, 4 of these patients had not undergone the proper diagnostic testing protocol. In 7 of the patients who received a diagnosis of pulmonary embolism, the physician had performed more diagnostic tests than were called for by the algorithm. In 759 of the 849 patients in whom pulmonary embolism was not found on initial evaluation, the diagnostic protocol was followed correctly. Only 1 (0.1% [CI, 0.0% to 0.7%]) of these 759 patients developed thromboembolic events during follow-up. Of the 437 patients with a negative D-dimer result and low clinical probability, only 1 developed pulmonary embolism during follow-up; thus, the negative predictive value for the com-

bined strategy of using the clinical model with D-dimer testing in these patients was 99.5% (CI, 99.1% to 100%). CONCLUSION: Managing patients for suspected pulmonary embolism on the basis of pretest probability and D-dimer result is safe and decreases the need for diagnostic imaging.

Tobacco Use and Skin Disease—Vander Straten M, Carrasco D, Paterson MS, McCrary ML, Meyer DJ, Tyring SK. South Med J 2001 Jun;94(6):621-634.

BACKGROUND: The primary objective of this review is to evaluate the mucocutaneous manifestations of tobacco use. METHODS: Computerized literature searches were conducted for English language articles related to skin/mucous membrane disease and use of tobacco. The primary criterion for assessing data quality and validity was the demonstration of a causal relationship between tobacco use and skin/mucous membrane disease. RESULTS: This review of the literature shows that a number of disorders and diseases of the skin and mucous membranes are related to tobacco use. CONCLUSIONS: Since millions of persons use tobacco despite its well publicized relationship to increased mortality, knowledge of the mucocutaneous morbidity associated with tobacco use may help physicians in counseling their patients.

Changes in Arterial Oxygen Tension when Weaning Neonates from Inhaled Nitric Oxide—Sokol GM, Fineberg NS, Wright LL, Ehrenkranz RA. Pediatr Pulmonol 2001 Jul;32(1):14-19.

We set out to evaluate changes in arterial oxygen tension (P_{aO_2}) when weaning neonates from inhaled nitric oxide (INO). We reviewed the records of 505 prospectively collected INO weaning attempts on 84 neonates with hypoxic respiratory failure. P_{aO_2} values before and 30 min after weaning attempts were recorded. Relationships between change in P_{aO_2} and decreases in INO concentrations were investigated using regression analysis and ANOVA. P_{aO_2} decreased (-18.7 ± 1.8 torr; $p < 0.001$); when weaning INO. A stepwise decline in P_{aO_2} was observed weaning INO from 40 ppm. The greatest decline occurred when INO was discontinued (-42.1 ± 4.1 torr). Forward stepwise multiple regression using variables with significant relationships to the decline in P_{aO_2} identified the specific dose reduction 7($p < 0.001$), the prewean P_{aO_2} ($p < 0.001$), and surfactant therapy ($p = 0.018$) as the variables best describing the change in P_{aO_2} ($p = 0.004$, $r = 0.51$). In conclusion, a graded decline in P_{aO_2} occurs when reducing INO. INO should be weaned to less than 1 ppm before discontinuing its use. Prior surfactant treatment appears to enhance the oxygenation reserve when weaning INO.

Asthma in Preschool Children: Prevalence and Risk Factors—Haby MM, Peat JK, Marks GB, Woolcock AJ, Leeder SR. Thorax 2001 Aug;56(8):589-595.

BACKGROUND: The prevalence of asthma in children has increased in many countries over recent years. To plan effective interventions to reverse this trend we need a better understanding of the risk factors for asthma in early life. This study was undertaken to measure the prevalence of, and risk factors for, asthma in preschool children. METHODS: Parents of children aged 3-5 years living in two cities (Lismore, n=383; Wagga Wagga, n=591) in New South Wales, Australia were surveyed by questionnaire to ascertain the presence of asthma and various proposed risk factors for asthma in their children. Recent asthma was defined as ever having been diagnosed with asthma and having cough or wheeze in the last 12 months and having used an asthma medication in the last 12 months. Atopy was measured by skin prick tests to six common allergens. RESULTS: The prevalence of recent asthma was 22% in Lismore and 18% in Wagga Wagga. Factors which increased the risk of recent asthma were: atopy (odds ratio (OR) 2.35, 95% CI 1.49 to 3.72), having a parent with a history of asthma (OR 2.05, 95% CI 1.34 to 3.16), having had a serious respiratory infection in the first 2 years of life (OR 1.93, 95% CI 1.25 to 2.99), and a high dietary intake of polyunsaturated fats (OR 2.03, 95% CI 1.15 to 3.60). Breast feeding (OR 0.41, 95% CI 0.22 to 0.74) and having three or more older siblings (OR 0.16, 95% CI 0.04 to 0.71) decreased the risk of recent asthma. CONCLUSIONS: Of the factors tested, those that have the greatest potential to be modified to reduce the risk of asthma are breast feeding and consumption of polyunsaturated fats.

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Bedside Tracer Gas Technique Accurately Predicts Outcome in Aspiration of Spontaneous Pneumothorax—Kiely DG, Ansari S, Davey WA, Mahadevan V, Taylor GJ, Seaton D. Thorax 2001 Aug;56(8):617-621.

BACKGROUND: There is no technique in general use that reliably predicts the outcome of manual aspiration of spontaneous pneumothorax. We have hypothesised that the absence of a pleural leak at the time of aspiration will identify a group of patients in whom immediate discharge is unlikely to be complicated by early lung re-collapse and have tested this hypothesis by using a simple bedside tracer gas technique. METHODS: Eighty four episodes of primary spontaneous pneumothorax and 35 episodes of secondary spontaneous pneumothorax were studied prospectively. Patients breathed air containing a tracer (propellant gas from a pressurised metered dose inhaler) while the pneumothorax was aspirated percutaneously. Tracer gas in the aspirate was detected at the bedside using a portable flame ioniser and episodes were categorised as tracer gas positive (>1 part per million of tracer gas) or negative. The presence of tracer gas was taken to imply a persistent pleural leak. Failure of manual aspiration and the need for a further intervention was based on chest radiographic appearances showing either failure of the lung to re-expand or re-collapse following initial re-expansion. RESULTS: A negative tracer gas test alone implied that manual aspiration would be successful in the treatment of 93% of episodes of primary spontaneous pneumothorax ($p < 0.001$) and in 86% of episodes of secondary spontaneous pneumoth-

orax ($p = 0.01$). A positive test implied that manual aspiration would either fail to re-expand the lung or that early re-collapse would occur despite initial re-expansion in 66% of episodes of primary spontaneous pneumothorax and 71% of episodes of secondary spontaneous pneumothorax. Lung re-inflation on the chest radiograph taken immediately after aspiration was a poor predictor of successful aspiration, with lung re-collapse occurring in 34% of episodes by the following day such that a further intervention was required. CONCLUSIONS: National guidelines currently recommend immediate discharge of patients with primary spontaneous pneumothorax based primarily on the outcome of the post-aspiration chest radiograph which we have shown to be a poor predictor of early lung re-collapse. Using a simple bedside test in combination with the post-aspiration chest radiograph, we can predict with high accuracy the success of aspiration in achieving sustained lung re-inflation, thereby identifying patients with primary spontaneous pneumothorax who can be safely and immediately discharged home and those who should be observed overnight because of a significant risk of re-collapse, with an estimated re-admission rate of 1%.

Comparison of the Standard Gamble, Rating Scale, AQLQ and SF-36 for Measuring Quality of Life in Asthma—Juniper EF, Norman GR, Cox FM, Roberts JN. Eur Respir J 2001 Jul;18(1):38-44.

With interest in health economics growing, there is a demand for valid methods for measuring health-related quality of life (HRQL) in asthma using utilities. The aims of this study were to develop disease-specific versions of the standard gamble and rating scale, to compare their measurement properties with those of the Asthma Quality of Life Questionnaire (AQLQ) and the Medical Outcomes Survey Short-Form 36 (SF-36), as well as to determine their validity for assessing asthma-specific quality of life. Forty adults with symptomatic asthma participated in a 9-week observational study. Participants completed the standard gamble, rating scale, AQLQ, SF-36 and other measures of clinical asthma status at baseline and after 1, 5 and 9 weeks. In patients whose asthma was stable between assessments, reliability was good for the rating scale (intraclass correlation coefficient (ICC)=0.89) and the AQLQ (ICC=0.95) but more modest for the SF-36 mental score (ICC=0.68), SF-36 physical score (ICC=0.65) and standard gamble (ICC=0.59). The responsiveness index was highest in the AQLQ (1.35), followed by the rating scale (0.74), the physical score of the SF-36 (0.61) and the standard gamble (0.31). Construct validity (correlation with other indices of health status) was strongest for the AQLQ and the rating scale. In conclusion, both the disease-specific rating scale and the Asthma Quality of Life Questionnaire have strong measurement properties for measuring asthma-specific quality of life; the Short-Form 36 health survey physical summary score has more modest properties. Although the disease-specific standard gamble has acceptable discriminative properties, its evaluative properties are too inadequate for it to be used in cost/utility analyses. Poor correlation between the standard gamble and the rating scale indicates that utilities cannot be derived from rating scale data.

Ventilatory and Cerebrovascular Responses in Normocapnic and Hypercapnic COPD Patients—Van de Ven MJ, Colier WN, Van der Sluijs MC, Kersten BT, Oeseburg B, Folgering H. Eur Respir J 2001 Jul;18(1):61-68.

This study investigated the hypothesis that hypercapnia in some chronic obstructive pulmonary disease (COPD) patients may be related to a high cerebrovascular response to carbon dioxide (CO_2). The relationship between responses of ventilation and of cerebral blood volume (CBV) to acute changes in carbon dioxide tension in arterial blood (P_{aCO_2}) was measured in 17 chronic hypercapnic ($P_{aCO_2} > 6.0$ kPa) and 16 normocapnic ($P_{aCO_2} \leq 6.0$ kPa) COPD patients, who were matched for degree of airway obstruction (forced expiratory volume in one second 27% predicted). Results were compared with 15 age-matched healthy subjects. CBV was measured using near infrared spectroscopy during normo- and hypercapnia and related to inspired minute ventilation ($\dot{V}_I$) and mouth occlusion pressure ($P_{0.1}$). Hypercapnia (end-tidal pressure of carbon dioxide (ΔP_{ETCO_2}) > 1 kPa) was induced by giving adequate amounts of CO_2 in the inspired air. During normocapnia, CBV (mL x 100 g^{-1}) was 2.41 ± 0.66 and 2.90 ± 0.60 (mean ± SD) in the normocapnic and chronic hypercapnic patients, respectively, which was significantly lower compared to healthy subjects (3.53 ± 0.77). All slopes of CO_2 responsiveness ($\Delta CBV/\Delta P_{aCO_2}$, $\Delta \dot{V}_I/\Delta P_{aCO_2}$, $\Delta P_{0.1}/\Delta P_{aCO_2}$) were significantly lower in both COPD groups relative to healthy subjects, but were not significantly different between the COPD groups. A poor but positive correlation between ventilatory and cerebrovascular CO_2 responsiveness ($\Delta CBV/\Delta P_{aCO_2}$ and $\Delta \dot{V}_I/\Delta P_{aCO_2}$) was found in COPD patients and healthy subjects. The findings do not support the hypothesis of abnormal cerebrovascular responses to carbon dioxide in hypercapnic chronic obstructive pulmonary disease patients.

Health-Related Quality of Life in COPD: Why Both Disease-Specific and Generic Measures Should Be Used—Engstrom CP, Persson LO, Larsson S, Sullivan M. Eur Respir J 2001 Jul;18(1):69-76.

Although research has consistently demonstrated that chronic obstructive pulmonary disease (COPD) impairs health-related quality of life (HRQL), little agreement has been evidenced regarding the factors identified as contributing to impaired HRQL. The aim was to study such factors using well established generic and specific HRQL instruments. The patients (n=68) were stratified by forced expiratory volume in one second (FEV_1) to represent a wide range of disease severity. Pulmonary function, blood gases and 6-min walking distance test (6MWD) were assessed. HRQL instruments included: St George's Respiratory Questionnaire (SGRQ), Sickness Impact Profile (SIP), Hospital Anxiety and Depression Scale and Mood Adjective Check List. The strength of the impact of COPD on HRQL was represented along a continuum ranging from lung function, functional status (physical and psychosocial) to well-being. Although correlations between FEV_1 versus SGRQ total and SIP overall scores (r=-0.42 and -0.32) were stronger than previously reported, multiple regression analyses showed that lung function contributed little to the variance when dyspnoea-related limitation, depression scores and 6MWD were included in the models. These three factors were important to varying degrees along the whole range of HRQL. Physiological, functional and psychosocial consequences of chronic obstructive pulmonary disease are only poorly to moderately related to each other. The present study concludes that a comprehensive assessment of the effects of chronic obstructive pulmonary disease requires a battery of instruments that not only tap the disease-specific effects, but also the overall burden of the disease on everyday functioning and emotional wellbeing.

Partial Liquid Ventilation Improves Lung Function in Ventilation-Induced Lung Injury—Vazquez de Anda GF, Lachmann RA, Verbrugge SJ, Gommers D, Haitsma JJ, Lachmann B. Eur Respir J 2001 Jul;18(1):93-99.

Disturbances in lung function and lung mechanics are present after ventilation with high peak inspiratory pressures (PIP) and low levels of positive end-expiratory pressure (PEEP). Therefore, the authors investigated whether partial liquid ventilation can re-establish lung function after ventilation-induced lung injury. Adult rats were exposed to high PIP without PEEP for 20 min. Thereafter, the animals were randomly divided into five groups. The first group was killed immediately after randomization and used as an untreated control. The second group received only sham treatment and ventilation, and three groups received treatment with perfluorocarbon (10 mL x kg^{-1}, 20 mL x kg^{-1}, and 20 mL x kg^{-1} plus an additional 5 mL x kg^{-1} after 1 h). The four groups were maintained on mechanical ventilation for a further 2-h observation period. Blood gases, lung mechanics, total protein concentration, minimal surface tension, and small/large surfactant aggregates ratio were determined. The results show that in ventilation-induced lung injury, partial liquid ventilation with different amounts of perflubron improves gas exchange and pulmonary function, when compared to a group of animals treated with standard respiratory care. These effects have been observed despite the presence of a high intra-alveolar protein concentration, especially in those groups treated with 10 and 20 mL of perflubron. The data suggest that replacement of perfluorocarbon, lost over time, is crucial to maintain the constant effects of partial liquid ventilation.

Bringing Scientific Evidence to the Ventilator Weaning and Discontinuation Process: Evidence-Based Practice Guidelines

Mechanical ventilatory support is a critical life sustaining modality for patients in respiratory failure. However, as the need for this support resolves, it is imperative that clinical attention be turned to the discontinuation process. Unnecessary prolongation of support is clearly associated with excess morbidity and costs. Clinical aggressiveness in the discontinuation process, however, must be tempered by the realization that premature removal of support can also induce patient harm.

Many questions face clinicians in dealing with the issue of discontinuing mechanical ventilatory support. Among these are: What are the reasons the patient required initiation/prolongation of mechanical ventilatory support, and is the patient stabilizing/reversing? What clinical signs/tests indicate that ventilator discontinuation is possible or likely? What is the best way to manage a patient who is not ready for discontinuation but who is improving? What is the role of tracheotomy? What is the role of specialized units or centers for the ventilator withdrawal process?

To help answer these questions using evidence-based approaches, 2 projects were initiated in 1999 that culminated in the development of the evidence-based practice guidelines published in this issue of Respiratory Care.

See the Special Article on Page 69

The first project was the McMaster University Evidence-Based Review of Weaning from Mechanical Ventilation.[1] This effort was sponsored by the United States Agency for Health Care Policy and Research (AHCPR) and initially searched over 5,000 citations from several databases to come up with 154 studies deemed suitable for an evidence-based report. The final report for the AHCPR contained 15 comprehensive data tables and 15 appendices describing the details of the analyses. The second project was the establishment of a task force by the American Association for Respiratory Care (AARC), the American College of Chest Physicians (ACCP), and the Society for Critical Care Medicine (SCCM) to create a set of evidence-based clinical practice guidelines for ventilator weaning and discontinuation. This task force relied heavily on the McMaster AHCPR document for evidence-based material but also sought additional expertise for issues not specifically addressed in this report. The task force used a consensus process to formulate recommendations based on evidence when possible and used expert opinion when such evidence was lacking.

Although many useful recommendations are contained in these guidelines, 4 particular issues are worth additional emphasis. First, the task force reviewed a substantial body of data that indicated how poorly clinicians do at assessing ventilator discontinuation potential, especially in the patient deemed "ventilator dependent" for more than several days. This finding underscores the need for more focused assessment strategies for such patients. Second, the available data are quite clear in showing that the best indicator of ventilator discontinuation potential is the clinical assessment (eg, respiratory pattern, cardiovascular response, comfort/anxiety, oxygenation) of patients during 30–120 minute spontaneous breathing trials (SBTs). More complex "weaning parameters" focused on physiologic measurements—such as muscle strength, respiratory system mechanics, metabolic parameters, and work of breathing—add little to the assessment of individual patients for discontinuation potential. Third, for patients failing a SBT, there are virtually no data showing that progressive ventilator reduction strategies between every-24-hour SBTs offer any benefit in shortening the duration of ventilatory support. Put another way, more cost-effective use of health care professionals would seem better attained by searching for reversible causes of respiratory failure rather than ritualistic attempts at gradual reductions in ventilator settings. As long as SBTs are performed every 24 hours, stable, comfortable, assisted forms of ventilatory support aimed at providing a non-fatiguing breathing load seem all that is necessary. Finally, the evidence is very clear that nonphysician health care professionals (especially respiratory therapists) can effectively achieve all of these goals when operating under well designed ventilator management protocols.

Now that the 1999–2001 projects are complete, it is hoped that this comprehensive work will provide clinicians with solid evidence-based recommendations for clinical practice. In addition, it is also hoped that this report

will show investigators where the important "holes" in our knowledge are, so that properly designed studies can be performed in the future.

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A Survey of Albuterol Administration Practices in Intubated Patients in the Neonatal Intensive Care Unit

Julie Ballard RRT, Ralph A Lugo PharmD, and John W Salyer RRT MBA FAARC

INTRODUCTION: Aerosolized albuterol is commonly used in the treatment of neonatal respiratory illnesses. Clinical and in vitro studies have identified numerous factors that affect aerosol drug delivery during neonatal mechanical ventilation, including the choice of metered-dose inhaler (MDI) or nebulizer, the use of a holding chamber, time between actuations, the volume of nebulized solution, and the position and placement of the nebulizer or MDI. Because there is no consensus on the optimal method of administration, there is probably substantial variability among institutions in how aerosolized albuterol is administered to mechanically ventilated infants in the neonatal intensive care unit (NICU). **OBJECTIVE:** Survey academic medical centers in the United States regarding their practices of administering aerosolized albuterol to intubated newborns in the NICU. **METHODS:** A survey instrument was developed that queried 18 aspects of albuterol administration in mechanically ventilated infants, including the frequency of MDI and nebulizer use, the average and maximum dose, the time between MDI actuations and following the final actuation, the use of a holding chamber, and the placement location of the holding chamber or nebulizer. Respiratory therapists and respiratory therapy managers having direct knowledge of neonatal clinical practices in their neonatal fellowship program NICUs were surveyed via telephone. Those who did not respond via telephone were surveyed via fax. **RESULTS:** Eighty institutions were surveyed and there were 68 respondents (85% response rate). Responders averaged 35 ± 13 NICU beds and 11 ± 5 ventilators/d. Nineteen percent of the respondents reported administering albuterol via MDI 100% of the time; 22% use MDIs 75–99% of the time; 9% use MDIs 50–74% of the time; 4% use MDIs 25–49% of the time; and 43% never use MDIs to deliver albuterol. The average dose via MDI was: 1 puff: 30%; 2 puffs: 65%; and 4 puffs: 5%. The maximum dose via MDI was: 2 puffs: 30%; 3 puffs: 14%; 4 puffs: 36%; 6 puffs: 11%; and 8 puffs: 6%. Thirty-one percent of the respondents place the holding chamber in-line with the ventilator circuit, 56% administer the aerosol via manual ventilation, and 13% use both methods. Fifty-six percent place the in-line holding chamber between the endotracheal tube and ventilator circuit, and the other 44% place the in-line holding chamber in the inspiratory limb. The time between MDI actuations depended on whether the holding chamber was placed in-line or the aerosol was administered via manual ventilation (MV): ≤ 0.5 min: 18% in-line and 28% MV; 1 min: 47% in-line and 43% MV; 2 min: 6% in-line and 4% MV; 3 min: 6% in-line and 0% MV. Eighty-three percent of respondents indicated that dead space introduced by a holding chamber/spacer was not a concern. Forty-three percent use nebulizers exclusively to administer albuterol to mechanically ventilated patients. Seventy-four percent of centers that nebulize albuterol use a dose of 1.25–2.5 mg. Eighty-eight percent of the surveyed institutions place nebulizers in-line with the ventilator circuit, and the other 12% use manual ventilation to administer the nebulized aerosol. Of those that use in-line nebulization, 95% place the nebulizer in the inspiratory limb of the circuit, and the other 5% place the nebulizer between the endotracheal tube and circuit Y-piece. Among centers that place the nebulizer in the inspiratory limb, 52% place it adjacent to the circuit Y-piece, 36% place it midway upstream in the inspiratory limb, and 12% place it near the humidifier. **CONCLUSION:** There is substantial variability among NICUs in albuterol administration to mechanically ventilated infants, with the majority of institutions now administering albuterol via MDI. *Key words: albuterol, adrenergic beta agonists, administration, inhalation, nebulizer, ventilator, metered-dose inhaler, MDI, neonatal.* [Respir Care 2002;47(1):31–38]

Introduction

Albuterol is commonly used in the treatment of neonatal respiratory illnesses such as bronchopulmonary dysplasia. In mechanically ventilated infants, β agonists decrease respiratory system resistance and P_{CO_2}, and increase oxygenation, tidal volume, and respiratory system compliance.[1–4] One goal in treating mechanically ventilated newborns with aerosolized medication is to optimize drug delivery through the ventilator circuit and to the lungs. Clinical and in vitro studies have identified numerous factors that affect aerosol drug delivery during neonatal mechanical ventilation, including the choice of metered-dose inhaler (MDI) or nebulizer, the use of a holding chamber, time between actuations, the volume of solution nebulized, and the position and placement of the nebulizer or MDI. Despite a substantial amount of research on this subject, there remains a lack of consensus on the optimal method of administering albuterol, and there is probably substantial variability among neonatal intensive care units (NICUs) in the administration of albuterol.

Historically, wet nebulization has been the most common method of administering albuterol to mechanically ventilated neonates. In a 1996 survey (unpublished data), we found that 85% of NICUs never used MDIs. That may have been because of the perceived risks of using MDIs and spacers with small babies. In 1991, the American Respiratory Care Foundation and the American Association for Respiratory Care published an Aerosol Consensus Statement, which stated that "in-line MDIs should not be used in mechanically ventilated infants with a tidal volume less than 100 mL because of the potential hypoxic mixture that results from the volume of chlorofluorocarbon gas released from the MDI."[5] A more recent statement (1995) by the American Association for Respiratory Care regarding aerosol delivery to neonatal and pediatric patients warns that "additional dead-space volume can occur when a spacer device is placed at the end of an artificial airway."[6] Despite these warnings, it appears that the use of MDIs in mechanically ventilated patients has increased in recent years. This may be because of the several potential advantages MDIs have over nebulization, including more efficient drug delivery to the lung,[7] no required adjustment of ventilator settings, less risk of aerosolized bacterial contamination associated with nebulizer treatments,[8–10] less personnel time, and lower hospital costs and charges.[11–15] Our literature search found no data on how clinicians are administering aerosolized albuterol to mechanically ventilated neonates, nor recent information on the extent of MDI use in NICUs, so we surveyed NICUs at academic medical centers regarding their aerosolized albuterol administration practices with intubated NICU patients.

Methods

A survey instrument (Table 1) was developed by an expert panel (nurse, respiratory therapists, and clinical pharmacist) to measure 18 aspects of albuterol administration to mechanically ventilated neonates, including (1) the frequency of MDI versus nebulizer use in administering albuterol, (2) the average and maximum dose, (3) the time between MDI actuations, (4) the frequency of holding chamber use, and (5) placement of the holding chamber or nebulizer (in-line with the ventilator circuit or via manual ventilation with a resuscitation bag). The survey was conducted in 1999. All academic medical centers in the United States that have neonatal/perinatal fellowship programs were contacted via telephone.[16] Participation was requested from respiratory therapists or respiratory care managers having direct knowledge of neonatal clinical practices at their facilities. If telephone contact with the appropriate individual was unsuccessful after numerous attempts, the survey was faxed. Descriptive statistics are used to report results.

Results

There were 68 respondents from the 80 hospitals eligible for participation (85% response rate). Sixty-seven centers were successfully surveyed via telephone and 1 center responded via fax. Figure 1 illustrates the regions of the surveyed hospitals, in accordance with the regions used by the American Hospital Association. Respondents averaged 35 ± 13 NICU beds and 11 ± 5 ventilators per day. All NICUs were Level III. Table 1 reports the response rate for each question.

Albuterol Administered via MDI

Fifty-seven percent of the respondents reported administering albuterol to ventilated neonates via MDI. Table 2 outlines the frequency of MDI use. Table 3 describes the average and maximum number of actuations of albuterol. Of those that administer albuterol to ventilated neonates

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A version of this report was presented by Julie Ballard RRT at the OPEN FORUM of the 45th International Respiratory Congress, Las Vegas, Nevada, December 16, 1999.

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Table 1. Survey Questions Regarding Albuterol Administration to Mechanically Ventilated Neonates in Hospitals with Neonatal Fellowship Programs*

Questions 1–10 refer to albuterol administration to mechanically ventilated neonates.

1. What percent of albuterol is administered by nebulizer? (100% response)
2. What percent of albuterol is administered by metered-dose inhaler (MDI)? (100% response)
3. What is the average dose of albuterol administered by nebulizer? (89% response)
4. What is the maximum dose of albuterol administered by nebulizer? (30% response)
5. What is the average number of actuations when you administer albuterol by MDI? (95% response)
6. What is the maximum number of actuations when you administer albuterol by MDI? (92% response)
7. Do you use a holding chamber/spacer to administer the MDI? (97% response)
8. If you use a holding chamber/spacer, would you consider it large-volume spacer or a small-volume MDI adapter? (61% response)
9. When you administer the MDI, do you place the holding chamber/spacer adapter in-line with the ventilator circuit or handbag it in (ie, manual ventilation)? (100% response)
10. Are you concerned about the effects of the holding chamber's dead space when administering MDI to an intubated neonatal patient? (92% response)

Questions 11–13 refer to holding chamber placement in-line with the ventilator circuit.

11. Do you put the holding chamber/spacer or MDI adapter between the endotracheal tube and ventilator circuit, in the inspiratory limb of the ventilator circuit, or at another location? (94% response)
12. How long do you wait between actuations (in either seconds or number of breaths)? (100% response)
13. How long do you wait (in either seconds or breaths) before removing the spacer after the last actuation? (94% response)

Questions 14–16 refer to MDI administration by manual ventilation.

14. What is the wait time in between actuations in either seconds or breaths? (93% response)
15. What is the wait time after the last actuation in either seconds or breaths? (93% response)
16. Do you put the patient back on the ventilator in between actuations? (89% response)

Questions 17–18 refer to albuterol administration by nebulization.

17. Is the nebulizer placed at the humidifier, midway down the inspiratory limb of the circuit, in the inspiratory limb of the circuit next to the Y-piece, between the circuit and the endotracheal tube; administered with a manual resuscitator; or other? (93% response)
18. What is the final volume placed in the nebulizer cup (in mL), including albuterol and diluent? (91% response)

Questions 19–21 refer to NICU demographics.

19. How many neonatal ICU beds in your NICU? (100% response)
20. What is the level of your NICU? (100% response)
21. What is the average number of ventilators in the NICU per day? (100% response)

*The maximum number of possible respondents varied with each question. The percent response reported parenthetically after each question was determined from the number of centers answering the question and the total number of possible respondents for that question.
ICU = intensive care unit
NICU = neonatal intensive care unit

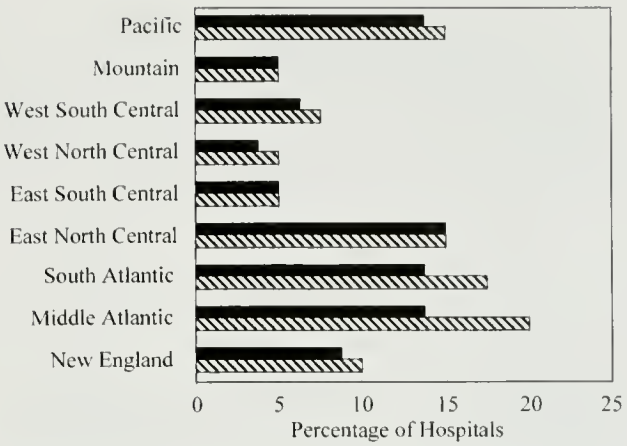

Fig. 1. Regions surveyed (in accordance with those used by the American Hospital Association). Pacific: Alaska, California, Hawaii, Oregon, and Washington. Mountain: Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah, and Wyoming. West North Central: Iowa, Kansas, Minnesota, Missouri, Nebraska, North Dakota, and South Dakota. East North Central: Illinois, Indiana, Michigan, Ohio, and Wisconsin. West South Central: Arkansas, Louisiana, Oklahoma, and Texas. East South Central: Alabama, Kentucky, Mississippi, and Tennessee. South Atlantic: Delaware, Washington DC, Florida, Georgia, Maryland, North Carolina, South Carolina, Virginia, and West Virginia. Middle Atlantic: New Jersey, New York, and Pennsylvania. New England: Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, Vermont. Black bars represent respondents. Hatched bars represent all hospitals.

Table 2. Frequency of Albuterol Administration via MDI to Mechanically Ventilated Infants in the NICU

Albuterol Administered via MDI (% of the time)	Respondents (%) (n = 68)
100	19
75–99	22
50–74	9
25–49	4
1–24	3
0	43

MDI = metered-dose inhaler
NICU = neonatal intensive care unit

via MDI (n = 39), a holding chamber is used by 95% of respondents. The majority (74%) of centers that use holding chambers use the large-volume type rather than a small-volume MDI adapter. Of the respondents who administer albuterol via MDI with holding chamber, 56% do so via manual ventilation, 31% insert the holding chamber into the circuit (in-line), and the remainder (13%) use both methods. Among the centers that place the holding chamber in-line, 56% place it between the endotracheal tube (ETT) and circuit Y-piece, and the remainder (44%) place it in the inspiratory limb. Only 17% of respondents who use holding chambers were concerned about dead space.

Table 3. Average and Maximum Number of MDI Actuations per Dose of Albuterol Administered to Mechanically Ventilated Infants in the NICU

Average Number	Respondents (%) (n = 37)
1	30
2	65
3	0
4	5
6	0
8	0
40	0

Maximum Number	Respondents (%) (n = 36)
1	0
2	30
3	14
4	36
6	11
8	6
40	3

MDI = metered-dose inhaler
NICU = neonatal intensive care unit

Table 4 lists the time between actuations and the time before removing the holding chamber following the last actuation, for both in-line placement and administration via manual ventilation. Five centers reported that they leave the holding chamber in-line between treatments. One facility that uses manual ventilation to administer the MDI treatment reported placing the patient back on the ventilator between actuations.

Albuterol Administered via Nebulizer

Forty-three percent of respondents use only nebulizers to administer albuterol to mechanically ventilated neonates. One hospital reported instilling albuterol solution directly down the ETT. Table 5 lists the average and maximum doses. The majority of centers (88%) place nebulizers in-line, and the other 12% use manual ventilation to administer the nebulized aerosol. Of those that use in-line nebulization, 95% place the nebulizer in the inspiratory limb of the circuit, and the other 5% place the nebulizer between the ETT and the circuit Y-piece. Among centers that place the nebulizer in the inspiratory limb, 52% place it adjacent to the circuit Y-piece, 36% place it midway upstream in the inspiratory limb of the circuit, and 12% place it near the circuit humidifier. Table 6 reports the final nebulizer volume.

Discussion

A 1988 pattern-of-practice survey of 323 institutions found that aerosolized bronchodilators were administered to mechanically ventilated neonates exclusively via nebulizer.[17] Twenty-two percent administered the nebulized treatment via manual ventilation, 24% placed the nebulizer some distance upstream in the inspiratory limb, and 54% placed the nebulizer in-line at the patient Y-piece.

In a 1996 survey of 1,078 institutions, 85% of NICUs reported that MDIs were never used, 10% used MDIs occasionally, 3% used them frequently, and 2% used them very frequently (unpublished data).[18]

Our survey results indicate that MDI use for intubated NICU patients has increased substantially during the last 10 years. Fifty-seven percent of centers administer albuterol via MDI at least some of the time, and 19% reported using MDIs exclusively. Nevertheless, a sizable percentage of centers (43%) still use nebulizers exclusively. In addition, there is substantial variability in administration practices, including the dose of albuterol, the use of holding chambers, and the placement of the holding chamber or nebulizer.

The recent trend toward increased use of MDIs for mechanically ventilated patients is probably driven by the high efficiency of MDI drug delivery and the shorter administration time. The efficiency of MDI drug delivery in mechanically ventilated neonates ranges from 1.96–14.2% when a holding chamber is used.[7,19–21] In contrast, nebulized drug delivery to the intubated neonate is very inefficient: only 0.02–2.1% of the dose reaches the end of the ETT.[7,20–24] The inefficiency of nebulizers for ventilated neonates is probably due to many factors, including rainout in the ventilator circuit and loss of aerosol particles through the continuous flow of the neonatal circuit.[23] An additional advantage of MDIs is short administration time, which frees the respiratory therapist to perform other clinical activities.

Because of the reported inefficiency of nebulizing drugs, the usual nebulized dose of albuterol far exceeds the dose administered via MDI.[7] Though the optimal dose of nebulized albuterol in mechanically ventilated infants is not known, the literature indicates a wide range of effective doses. A study by Fok et al showed a 16.9–32.3% improvement of respiratory system resistance by aerosolizing just 200 μg of albuterol with a jet nebulizer.[25] Wilkie and Bryan reported that both compliance and resistance improved by nebulizing 0.02 mg/kg of albuterol.[26] Two studies indicated an 11–35% improvement in respiratory system compliance in ventilated premature infants by nebulizing 2.5–5.0 mg of albuterol solution.[2,27] We found that most neonatal centers nebulize 1.25–2.5 mg of albuterol. The Aerosol Consensus statement suggests that the number of actuations from an MDI should be titrated to

Table 4. Duration Measured in Minutes or Number of Breaths Between MDI Actuations, and Between the Last Actuation and Removal of the Holding Chamber in Mechanically Ventilated Neonates in the NICU

Duration	Between Actuations		After Last Actuation	
	In-Line (%) (n = 17)	Manual Ventilation (%) (n = 28)	In-Line (%) (n = 11)	Manual Ventilation (%) (n = 26)
Minutes				
≤ 0.5	18	28	9	27
1	47	43	55	23
2	6	4	9	4
3	6	0	0	0
Breaths				
≤5	17	18	18	38
6–10	0	7	9	8
30	6	0	0	0

MDI = metered-dose inhaler
NICU = neonatal intensive care unit
In-line = holding chamber inserted in-line with the ventilator circuit
Manual ventilation = aerosol administered via manual ventilation, using a resuscitation bag

Table 5. Average and Maximum Dose of Nebulized Albuterol Administered to Mechanically Ventilated Infants in the NICU

Average Dose	Respondents (%) (n = 50)
≤ 1 mg	22
1.25 mg	44
2.5 mg	30
0.05 mg/kg	2
0.10 mg/kg	2
0.20 mg/kg	0

Maximum Dose	Respondents (%) (n = 16)
≤ 1 mg	19
1.25 mg	19
2.5 mg	56
0.05 mg/kg	0
0.10 mg/kg	0
0.20 mg/kg	6

NICU = neonatal intensive care unit

Table 6. Total (Final) Volume of Solution Placed in the Nebulizer Cup*

Total Volume (mL)	Respondents (%) (n = 49)
2.0	6
2.5	21
3.0	65
4.0	2
5.0	4
6.0	2

*Including both albuterol solution and diluent (0.9% NaCl)

the patient's response,[5] but it is our experience that few clinicians do this with nebulized albuterol. Our survey reflects that practice: nearly all of the respondents reported that they do not exceed 2.5 mg per dose.

Appropriate placement of the nebulizer may substantially enhance drug delivery. Rau and Harwood reported that placing the nebulizer 5 inches from the circuit Y-piece nearly doubles drug delivery, compared to placement next to the Y-piece or administration via manual ventilation.[24] Watterberg et al reported no significant difference in drug delivery when the nebulizer was placed 60 cm upstream from the Y-piece, compared to placement at the ETT adapter.[28] The inconsistent results in the literature are perhaps reflected in the results of the present survey: 52% of centers that place the nebulizer in the inspiratory limb place it adjacent to the circuit Y-piece, 36% place it midway upstream in the inspiratory limb, and 12% place it near the circuit humidifier. Only 12% of respondents do not place the nebulizer in-line but rather use manual ventilation to administer the aerosol.

Studies have shown that the final fill volume of the nebulizer (ie, the total volume that is nebulized) influences the amount of drug nebulized. The majority (92%) of respondents use a final volume of 2.0–3.0 mL, which may be suboptimal. In one study, increasing the nebulizer volume fill from 2 mL to 5 mL more than doubled the amount of drug nebulized (20.5% vs 44.5%).[28] In another study, Clay et al reported that a 2 mL fill volume resulted in 50% nebulizer output, whereas a 4 mL fill volume increased nebulizer output to 60–80%.[29] Hess recommends a diluent

volume of 4 mL in order to minimize dead volume or the volume left after nebulization.[12]

The dose-response relationship of albuterol delivered via MDI has not been well studied in intubated infants. Several published studies have indicated that 90–200 μg of albuterol administered via MDI (1–2 actuations) is sufficient to improve pulmonary mechanics in the majority of intubated infants.[4,25,30,31] The present survey indicates that most centers (95%) administer a dose that is consistent with that body of literature. The Aerosol Consensus Statement suggests that the number of puffs from an MDI should not necessarily be reduced because of the patient's size, but should be titrated according to the patient's response.[5] The fact that 67% of respondents increase the dose to a maximum of 3–8 actuations seems to indicate that dose titration is occurring, although a maximum dose of 40 actuations per dose appears excessive, especially given the potential for cardiovascular adverse effects due to large doses of chlorofluorocarbon propellant from the MDI.[32,33]

Ninety-five percent of respondents reported using a holding chamber when administering albuterol via MDI. The use of a holding chamber permits the high-velocity drug/propellant mixture to slow, undergo evaporation, and produce particles in the respirable range. Approximately one fourth of these respondents use an adapter, and the remainder use the larger volume holding chambers (eg, AeroChamber or Aerosol Cloud Enhancer [ACE] spacer). Previous studies have found that holding chambers such as AeroChamber and ACE result in greater drug delivery to the end of the ETT than MDI adapters.[19,34] The small volume of the in-line MDI adapter may seem more attractive if one is placing it between the ETT and circuit Y-piece, since it adds less dead space than a 150–180 mL holding chamber. However, Lugo et al found in vitro that the potential for carbon dioxide accumulation with a small MDI adapter (19 mL internal volume) is similar to that with larger volume holding chambers (152 mL and 182 mL internal volume) placed between the ETT and Y-piece.[35]

Albuterol can be administered via MDI either by placing the holding chamber in-line or by disconnecting the patient from the ventilator and administering the aerosol via manual ventilation. Thirty-one percent of survey respondents administer the MDI in-line with the ventilator circuit, 56% use manual ventilation, and the rest use a combination of the 2 methods. In an in vitro neonatal ventilated lung model, albuterol delivery did not differ between those 2 methods.[7] The benefits of in-line administration include aerosol administration using heated and humidified gas. In addition, manual ventilation may produce inconsistent tidal volume and peak pressures, with the potential for morbidity, including pneumothoraces.

Of the respondents who place the holding chamber in-line with the ventilator circuit, 56% place it between the ETT and the circuit, and 44% place it in the inspiratory limb. Manufacturers often recommend placing the holding chamber in the inspiratory limb of an adult circuit immediately proximal to the circuit Y-piece. However, during pressure-limited, continuous-flow ventilation, that location may result in considerable drug loss through the expiratory limb.[36] Furthermore, it is often impractical to adapt holding chambers into small-diameter pediatric and neonatal circuits. By placing the holding chamber at the end of the ETT, the problem of continuous flow washing aerosol out the expiratory limb of the circuit is avoided. However, that placement also creates additional mechanical dead space and theoretically increases the risk of rebreathing carbon dioxide. It is of interest that 83% of respondents expressed no concern about dead space. In an in vitro study, Lugo et al reported that the accumulation of carbon dioxide in the ACE and AeroChamber-MV spacer is not likely to result in a clinically important increase in P_{CO_2}.[35] However clinical studies are necessary to corroborate that finding.

The time between actuations affects how long a holding chamber is left in-line or attached to the ETT. Since the addition of dead space may be of concern, some institutions have limited the time that holding chambers may be attached to the ETT. This results in short administration times. When the MDI is administered in-line, 18% of respondents wait ≤ 30 seconds between actuations and 17% wait ≤ 5 breaths before repeating the actuation. Among respondents who administer the MDI dose via manual ventilation, 28% wait ≤ 30 seconds and 18% wait ≤ 5 breaths between actuations. It is noteworthy that shortening the time between actuations and the total time that the holding chamber is in-line may have a detrimental effect on drug delivery. A recent in vitro study found significantly lower albuterol delivery with either 5 breaths (10 seconds) or 15 breaths (30 seconds) between actuations than with 30 breaths (60 seconds) between actuations.[37] Most in vivo and in vitro studies waited 30–60 seconds between actuations.[4,7,21,27,38] Among survey respondents, the most common waiting period was ≤ 60 seconds between actuations.

The present survey represents NICU practices in academic medical centers. We chose to survey institutions with fellowship programs as our sampling group because they typically represent centers where the latest developments in clinical practice are implemented. Since our response rate was high (85%), concern about sample bias is minimal. Thus, though our findings may not be completely representative of the neonatal community at large, we are confident that our survey accurately describes current practice at academic centers with neonatal fellowship programs. Furthermore, the homogeneity of the sample suggests that we probably underestimated the magnitude of practice variability among all NICUs.

Conclusions

Our survey indicates that MDI administration of albuterol to mechanically ventilated neonates has increased, but there remains substantial variability in the practice of bronchodilator administration in the neonatal population. Although numerous in vitro studies point to the greater efficiency of MDIs, additional clinical studies are warranted to evaluate the safety and efficacy of this method of drug administration.

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Evaluation of Work of Breathing in Spontaneously Breathing Animals During Partial Liquid Ventilation

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BACKGROUND: Partial liquid ventilation improves lung mechanics and gas exchange in paralyzed mechanically ventilated animals. OBJECTIVE: Examine the work of breathing (WOB) in a spontaneously breathing animal model during partial liquid ventilation with and without the use of pressure-support ventilation (PSV). METHODS: This was a prospective study including 6 lambs (mean weight 10.9 ± 1.3 kg). Baseline measurements, including total work of breathing (WOB_T), elastic work of breathing (WOB_E), and resistive work of breathing (WOB_R), were obtained using pressure-controlled synchronized intermittent mandatory ventilation with positive end-expiratory pressure of 5 cm H_2O at PSV levels of 0, 5, and 10 cm H_2O. The animals' lungs were filled with perflubron through an endotracheal tube, in 10–20 mL aliquots, until filled, approximately 30 mL/kg or functional residual capacity. Repeat measurements were obtained at 10 mL/kg, 20 mL/kg, and full. Perflubron was then allowed to evaporate from the lungs and repeat measurements were obtained 3 additional times, with at least a 1 hour separation between phases, for up to 7 hours after the lungs were filled. RESULTS: No differences were detected in WOB_T, WOB_R, or WOB_E between the gas-filled lung and the lung filled to functional residual capacity with perflubron. However, compared to the gas-filled lung, WOB_T and WOB_R were higher during the filling ($p < 0.05$) and evaporative phases ($p < 0.05$). The PSV level affected WOB. Work of breathing was least at PSV 10 cm H_2O. CONCLUSION: In this pilot study of healthy animals breathing spontaneously with perflubron-filled lungs, there was an acceptable amount of WOB, which decreased with the addition of PSV. However, WOB increased when the perflubron level was not maintained at functional residual capacity. *Key words: artificial ventilation, positive-pressure ventilation, inspiratory work, work of breathing, perflubron, positive end-expiratory pressure, liquid ventilation.* [Respir Care 2002;47(1):39–47]

Introduction

Partial liquid ventilation (PLV) with perflubron is intended as an adjunct to conventional mechanical ventilation in patients with acute respiratory distress syndrome and neonates with surfactant deficiency. It uses the combination of gas delivery (via a conventional mechanical ventilator) and perflubron-filled lungs. Perflubron is a biochemically inert perfluorochemical that has a low surface tension, a positive spreading coefficient, and a high solubility for oxygen and carbon dioxide. Numerous animal studies using lung disease models have shown that PLV can improve gas exchange[1–3] and lung mechanics such as pulmonary compliance.[4,5]

Promising Phase 1 data have been published for the use of PLV in premature infants,[6] and studies have been undertaken in both pediatric and adult patients with acute respiratory distress syndrome. Recently these studies have been completed, although questions persist concerning the ideal method for using mechanical ventilation during PLV. During the pediatric trial, patients were maintained on chemically induced paralysis for the duration of PLV treatment. However, during the adult trial, patients were al-

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lowed to breathe spontaneously during PLV. There are potential disadvantages to the use of neuromuscular blockade during mechanical ventilation, including prolonged muscle weakness, increased costs,[7] reduced cardiac output and blood pressure,[8] and lower functional residual capacity (FRC). Allowing patients to breathe spontaneously during PLV was discouraged during the pediatric trial because of a lack of data on PLV's effect on work of breathing (WOB). These concerns were based on the potential that WOB might be increased because of an increase in lung density during PLV. Also there may be concern about the patient's ability to trigger the ventilator across the liquid-gas interface during PLV. In contrast, there may be potential benefits from allowing the patient to breathe spontaneously during PLV. Previous studies with controlled mechanical ventilation found that neonatal patients breathing spontaneously in a patient-triggered mode (as compared to a non-patient-triggered mode) showed better gas exchange[9,10] and needed a lower level of mechanical support.[10,11] In addition, studies of neonates suggest that the use of patient-triggered ventilation increases the delivery of tidal volume (V_T),[12] shortens the length of mechanical ventilation,[13] and decreases WOB[14] by improving patient-ventilator synchrony.

Few data are available on the use of perflubron in spontaneously breathing animals[15] or spontaneous ventilation during PLV in mechanically ventilated animals.[16,17] No studies are available on the effects of pressure-support ventilation (PSV) during PLV in those animals.

To achieve normal ventilation, the body performs work, known as WOB, to overcome the elastic and frictional resistance of the lungs and chest wall. Total WOB (WOB_T) is composed of the elastic work of breathing (WOB_E) and the resistive work of breathing (WOB_R). Elastic WOB represents physiologic work that includes the work to expand the lungs and chest wall. Resistive WOB is considered a measure of imposed WOB and includes work caused by the breathing apparatus, such as the endotracheal tube (ETT), the breathing circuit, and the ventilator's demand-flow system. The objective of this pilot study was to examine WOB in a spontaneously breathing animal model during PLV with and without the use of PSV.

Methods

Animal Model

The Animal Use and Care Committee of the University of Arkansas for Medical Sciences approved this study. Animals were cared for in accordance with the standards for care and use of laboratory animals set forth by the University.

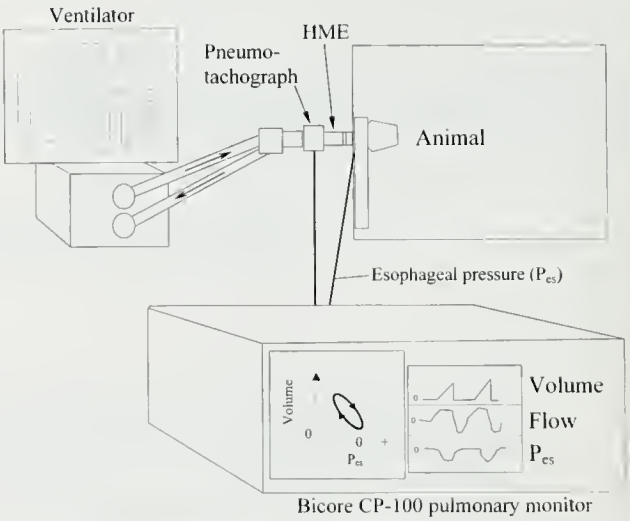

Fig. 1. Experimental setup. HME = heat-and-moisture exchanger.

Animal Model Preparation

Studies were performed on 6 young lambs. This number was based on a sample size analysis of an initial study measuring WOB with lambs on PSV of 5 cm H_2O and positive end-expiratory pressure (PEEP) of 5 cm H_2O, to provide 80% power to detect a 25% change in WOB between gas-filled and liquid-filled lungs, using preliminary data with a variance of 0.0007. Data were collected from 4–5-week-old Dorsett/Western Cross lambs. This animal model was selected because respiratory rate, V_T, and minute ventilation are comparable to those of pediatric patients.

Prior to the study day, vascular catheters were surgically placed via cutdown in the external jugular vein for drug administration and the internal carotid artery for blood pressure monitoring. After surgery, the animals were allowed to recover for 24–48 hours. On the study day, all animals were anesthetized (midazolam 0.25 mg/kg, propofol 0.3 mg/kg) and intubated with 5.0 mm inner diameter cuffed ETTs. Endotracheal tube location was confirmed by direct visualization with a bronchoscope, and the cuff was inflated to minimal occlusion pressure. After intubation, the animal was placed in prone position in a temperature-controlled chamber. Gas humidification was maintained by a heat-and-moisture exchanger (Portex Humid-Vent 600, Keene, New Hampshire) positioned on the ETT between the animal and the flow transducer (Fig. 1). Supplemental intravenous anesthesia (midazolam 0.3 mg/kg/h, propofol 10 mg/kg/h) was administered continuously via the external jugular venous catheter. The level of sedation was clinically monitored in order to maintain the animal in a light sleep, breathing spontaneously, arousable with interventions or stimulation, but easily returning to sleep. The animals' heart rate, respiratory breathing

frequency, arterial blood pressure, oxygen saturation (measured via pulse oximetry), and temperature were monitored continuously with a physiologic monitor (Siemens SC9000, Siemens Medical Systems, Danvers, Massachusetts).

Arterial blood gas values were obtained from all animals intermittently throughout the experiments. For the experimental design, the goal was to maintain pH $\geq$ 7.30 and $\leq$ 7.45.

Equipment Setup

The animals were sedated, intubated, and ventilated with a mechanical ventilator (Servo 300, Siemens Medical Systems, Danvers, Massachusetts) in the specific mode and settings of the study protocol. In each experiment the animal was ventilated in pressure-controlled synchronized intermittent mandatory ventilation (SIMV + PC) mode, with a set peak inspiratory pressure of 15 cm H_2O, respiratory rate of 5 breaths/min, PEEP of 5 cm H_2O, and randomly applied PSV levels of 0, 5, and 10 cm H_2O. All studies were conducted in the pediatric patient range, using flow-triggering, with the sensitivity set in the middle of the "green zone" on the ventilator.

Data Acquisition

Airway pressure and flow were measured at the proximal end of the ETT, using a differential flow transducer connected to the physiologic monitor (Fig. 1). The dead space within the flow transducer was 11 mL. Intrapleural pressure was inferred using a standard commercially manufactured esophageal catheter (SmartCath, VIASYS Healthcare, Yorba Linda, California), which was also connected to the pulmonary monitor. Esophageal balloon placement was confirmed by occlusion test.[18]

The BiCore CP-100 monitor records both measured and calculated respiratory variables, and we recorded airway pressure, esophageal pressure (P_{es}), and airway flow. Respiratory drive and respiratory time fraction (T_I/T_{TOT}) are calculated from those measurements. Diaphragmatic contraction is a response to stimulation from the neural drive, and respiratory drive is a measure of that drive.[19] It is defined as the airway occlusion pressure at the first 100 ms after airway closure, prior to the animal's conscious recognition of occlusion. This value represents work expenditure during the animal's initiated ventilator breath and, if elevated, it may represent an increase in work by the animal. Consequently, a low respiratory drive value may indicate when respiratory drive is blunted. Concurrently, T_I/T_{TOT} is a measure of endurance. T_I/T_{TOT} is a ratio and therefore has no unit of measure. If respiratory muscles fatigue, more time is spent in inspiration, thus T_I/T_{TOT} increases.[20]

Other calculated variables include respiratory rate, minute ventilation, the change in airway pressure, peak inspiratory pressure, intrinsic PEEP, change in P_{es}, and pressure-time product. The change in airway pressure is the maximum airway pressure measured during each breath minus the starting pressure required to move the same volume of gas. Peak inspiratory pressure is the maximum airway pressure reached from the start of inspiratory flow to the end of expiratory flow for each breath. Intrinsic PEEP is the difference between P_{es} at the end of expiration and P_{es} at the start of inspiratory flow. The change in P_{es} is the change in P_{es} as a result of ventilation. Pressure-time product is calculated by integrating P_{es} (end-expiratory P_{es} minus current P_{es}) and time for the duration of contraction of the respiratory muscles (current V_T/chest wall compliance). Optimal PSV decreases pressure-time product and varies directly with total lung resistance.[20]

In addition to the data from the pulmonary monitor, 10 consecutive breaths were collected for each phase of the experiment, for the measurement of WOB_T, WOB_R, and WOB_E, using the Campbell loop[21] as described by Blanch and Banner.[22] This procedure involved directly measuring chest wall compliance (C_{CW}) during mechanical inflation of the lungs and thorax while the animal was paralyzed. Chest wall compliance was directly measured by paralyzing the animal with succinylcholine (2 mg/kg) at the start of the experiment and at the beginning of each phase, while ensuring adequate respiratory rate and V_T with a control mode of mechanical ventilation. Analysis of the P_{es} and V_T loop using the Campbell-diagram software was integrated into the calculation of WOB to measure C_{CW}.[22] After C_{CW} is determined, P_{es}-V_T loops are then combined into a single composite graph, and WOB is measured from its planimetry by the pulmonary monitor. Immediately after paralysis for C_{CW} but before initiation of the experiment, a rest period was allowed for the animal to fully recover from the succinylcholine. We considered the animal fully recovered from the succinylcholine when it had returned to its normal baseline state (respiratory rate, pulse oximetry reading, and V_T, and was triggering the ventilator spontaneously).

Partial Liquid Ventilation

Perflubron (LiquiVent, Alliance Pharmaceutical, San Diego, California) was administered via the ETT in 10–20 mL aliquots, requiring an average of 2.5 hours for filling. A full lung was defined as administration of at least 30 mL/kg (or FRC), evidence of perflubron in the ETT on temporarily removing the animal from the ventilator, and a chest radiograph showing a full lung. No refilling was performed after a full lung was confirmed. Evaporation was allowed to occur, and repeat measurements were obtained 3 additional times, with at least a 1-hour separation

between phases, for up to 7 hours after filling. E1, E2, and E3 were intended to be 3 phases (separated by 1 hour) during evaporation of the perflubron. But the E1, E2, and E3 periods differed because of differences in the animals' cooperation and response, so we have described them as ranges of time rather than specific time periods. During all data collection phases of the experiment, the animals were maintained on SIMV + PC. Ventilation was maintained with SIMV + PC with PEEP of 5 cm H_2O and PSV of 5 cm H_2O between data collection phases.

Statistical Analysis

Data were analyzed as the averages of 10 breaths for each animal for a given liquid level and pressure support level. For WOB, since each animal was subjected to all levels of PLV and PSV, a mixed model analysis of variance was implemented, with animal as a random factor, level of PLV and pressure support as fixed effects, and the interaction of PLV level and pressure support as within-animal factors. This seemed to be the most effective way of controlling for experiment-wise error. However, since it could not be checked that this model satisfied assumptions, we also did pairwise *t* test comparisons and adjusted using a Bonferroni adjustment. Since the Bonferroni adjustment is conservative, we reported on all results that were consistent with the mixed model approach and were significant up to the $p < 0.1$ level after adjustment. For the other variables of interest, such as peak inspiratory pressure, the 2 filling levels were combined and averaged, as were the 3 evaporative stages. There were 4 liquid levels compared: gas ventilation (GV), filling, full, and evaporative phase. Within each pressure, pairwise comparisons for each variable of interest were carried out using a *t* test, and a Bonferroni adjustment was done for the 6 comparisons of the 4 levels. All analyses were conducted with commercially available statistical software (SPSS for Windows, SPSS Inc, Chicago, Illinois). A p value ≤ 0.05 was considered statistically significant, except during the Bonferroni adjustment.

Results

Mean weight of the lambs was 10.9 ± 1.3 kg. The mean study duration, from initiation of perflubron until the end of the study, was 9 hours (range 7–10 h). The instillation of perflubron took on average 147 minutes (range 104–198 min) to achieve lung filling and was well tolerated by all animals, with no evidence of hemodynamic or respiratory compromise. There were no episodes of arterial oxygen desaturation.

Arterial partial pressure of oxygen ranged from 331 to 549 mm Hg, pH ranged from 7.29 to 7.45, and arterial partial pressure of carbon dioxide ranged from 38 to 51 mm Hg. Arterial partial pressure of oxygen was higher during GV than during PLV for all animals ($p \leq 0.01$).

There was no evidence of intrinsic PEEP detected in any of the phases of the experiment.

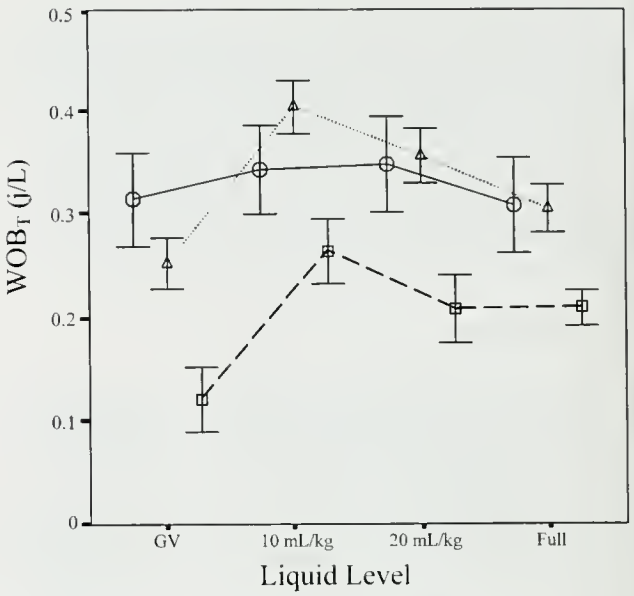

Fig. 2. Total work of breathing (WOB_T) at pressure-support ventilation (PSV) levels of 0 cm H_2O (circles), 5 cm H_2O (triangles), and 10 cm H_2O (squares) during gas ventilation (GV), during filling, and at full. Values are mean ± 2 SEM. There was no difference between GV and full or between GV and PSV of 20 mL/kg, but there was a difference between GV and 10 mL/kg. There was also a difference between PSV of 10 cm H_2O and the other 2 PSV levels ($p \leq 0.05$).

Partial Liquid Ventilation Filling

Figure 2 summarizes the WOB_T results for each of the filling phases of PLV at the various levels of PSV. Figure 3 summarizes the WOB_R results. Figure 4 summarizes the WOB_E results. For WOB_T and WOB_R there was only a liquid within-animal effect ($p \leq 0.003$, $p \leq 0.04$, respectively). This implies that as liquid levels changed, the change in WOB_T and WOB_R was dependent on the animal. This difference, due to changing liquid levels within the animals, was not between GV and full, but between GV and 10 mL/kg for WOB_T, and between GV and 10 mL/kg and 20 mL/kg for WOB_R. There was no difference between PSV of 0 and 5 cm H_2O, but PSV of 10 cm H_2O did differ ($p \leq 0.05$) from PSV of 5 cm H_2O for both WOB_T and WOB_R. The application of PSV of 10 cm H_2O lowered WOB_T and WOB_R. A liquid within-animal effect was also seen when WOB_E was measured ($p \leq 0.006$). For WOB_E, there were no differences when comparing GV to the filling or full phases; however, there was a difference between PSV levels ($p < 0.05$). Application of PSV decreased WOB_E.

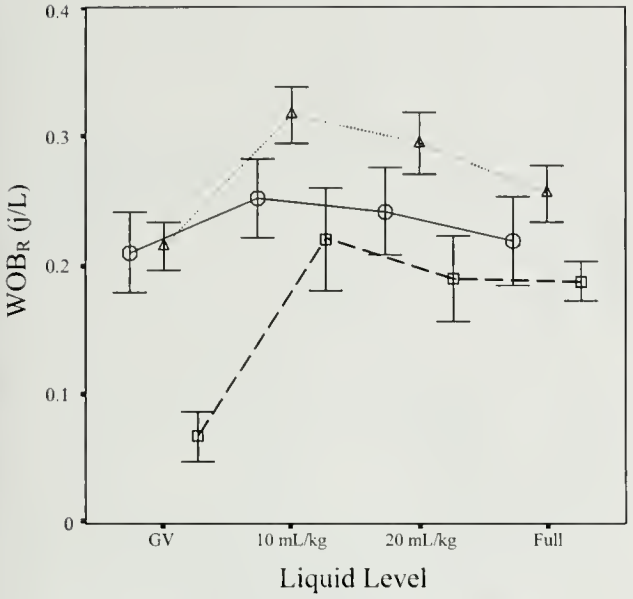

Fig. 3. Resistive work of breathing (WOB_R) at pressure-support ventilation (PSV) levels of 0 cm H_2O (circles), 5 cm H_2O (triangles), and 10 cm H_2O (squares) during gas ventilation (GV), during filling, and at full. Values are mean ± 2 SEM. There was no difference between GV and full, but there was a difference between GV and PSV of 10 and 20 mL/kg ($p \leq 0.05$). There was also a difference between PSV of 10 cm H_2O and the other 2 PSV levels ($p \leq 0.05$).

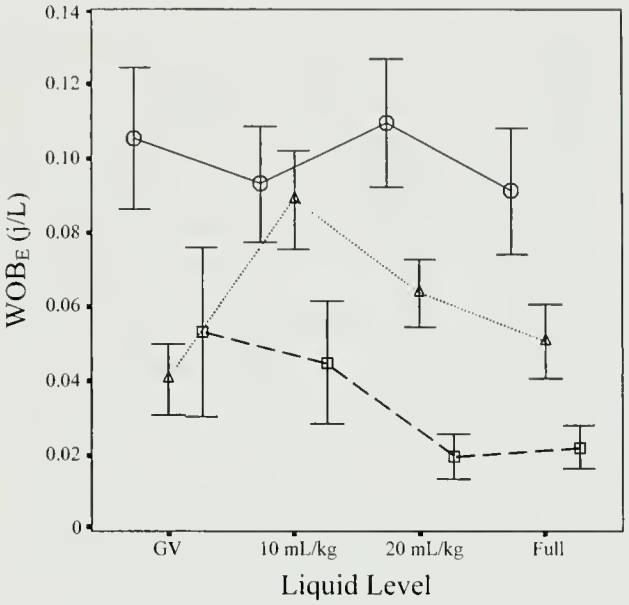

Fig. 4. Elastic work of breathing (WOB_E) at pressure-support ventilation (PSV) levels of 0 cm H_2O (circles), 5 cm H_2O (triangles), and 10 cm H_2O (squares) during gas ventilation (GV), during filling, and at full. Values are mean ± 2 SEM. There was no difference between GV and the other liquid levels. There was a difference between all PSV levels ($p \leq 0.05$).

Evaporative Phase

Figures 5–7 summarize the WOB_T, WOB_R, and WOB_E results for GV and each of the evaporative PLV phases at the various PSV levels. There was a liquid within-animal effect for WOB_T, WOB_R, and WOB_E ($p \leq 0.001$). For WOB_T there were no differences between GV and E1 or E2; however, within animals, there was a difference between GV and E3 ($p \leq 0.05$). For WOB_T there was a difference between PSV of 0 and 5 cm H_2O and PSV of 10 cm H_2O ($p \leq 0.05$). Application of PSV decreased WOB_T during all evaporative phases. For WOB_R there was no difference when comparing GV to E1, but there were differences within animals when comparing GV to E2 and E3 ($p \leq 0.05$). There were no differences when comparing PSV of 0 and 5 cm H_2O, but PSV of 10 cm H_2O differed from the other 2 PSV levels ($p \leq 0.05$). The application of PSV of 10 cm H_2O decreased WOB_R in the evaporative phases. For WOB_E there were no differences between GV and the evaporative phases. WOB_E appeared to decrease with the addition of PSV, but the differences were not significant.

Table 1 lists BiCore CP-100 data acquired during SIMV + PC with various PSV levels during the phases of lung filling and evaporation. Respiratory measurements are represented as mean values ± standard deviation for GV, filling, full, and evaporative phases for each PSV level. The filling phase values represent the average of the 10 and 20 mL/kg phases. The evaporative phase values represent the average of the E1, E2, and E3 phases. For all comparisons to GV there were no statistical differences.

Discussion

The principle finding of this pilot study is that if healthy animals are allowed to breathe spontaneously while being supported on PLV, they experience acceptable WOB levels. An additional finding was that the use of PSV decreases WOB in these animals. However, compared to GV, WOB increases if the perflubron level is not maintained at 30 mL/kg (or FRC).

The WOB levels measured during GV and PLV full were comparable to resistive work measured in previous studies in our laboratory.[23,24] In the present study we measured WOB_R and WOB_E and were therefore able to calculate WOB_T. Our results show that WOB decreased across all liquid levels with the addition of PSV but was not significantly different from GV once FRC was attained with perflubron. We expected that in a perflubron-filled lung the effects on WOB might be seen in resistive work or elastic work, or perhaps both. Effects on resistive work would be secondary to the presence of perflubron in the trachea and ETT, impeding the animal's ability to trigger the ventilator. In contrast, for elastic work the effects could be related to having a liquid-filled lung, thus necessitating

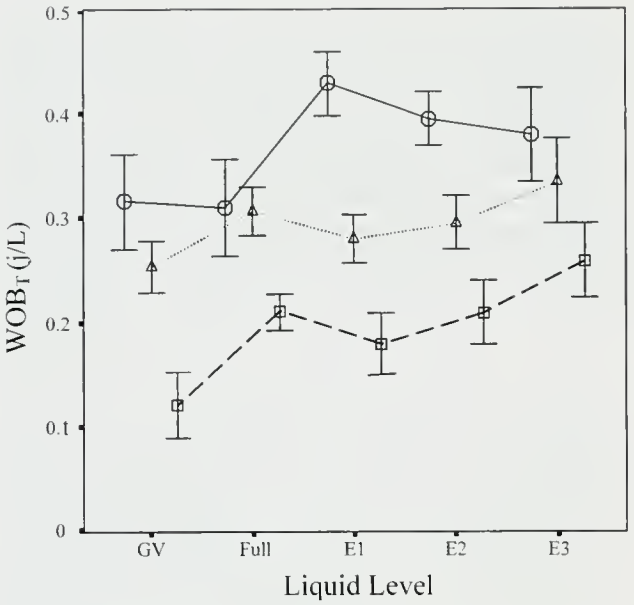

Fig. 5. Total work of breathing (WOB_T) at pressure-support ventilation (PSV) levels of 0 cm H_2O (circles), 5 cm H_2O (triangles), and 10 cm H_2O (squares) during gas ventilation (GV), at full, and during evaporation phases E1, E2, and E3. Values are mean ± 2 SEM. There was a difference between GV and E3 ($p \leq 0.05$). There was no difference between the evaporative levels. There was a difference between all PSV levels ($p \leq 0.05$).

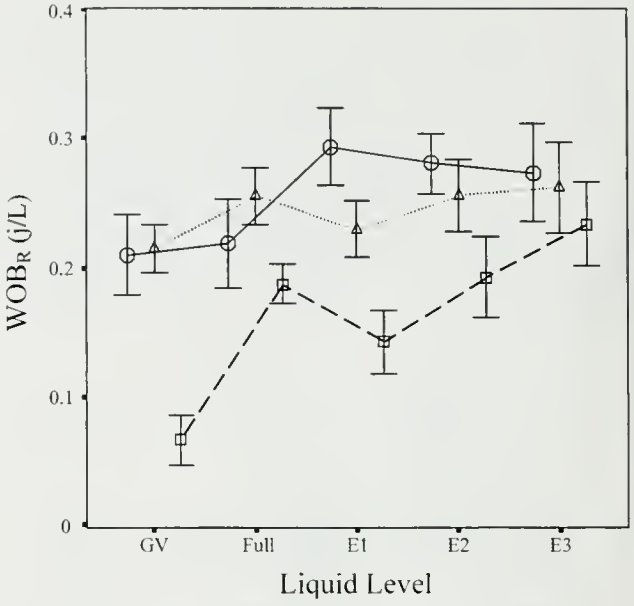

Fig. 6. Resistive work of breathing (WOB_R) at pressure-support ventilation (PSV) levels of 0 cm H_2O (circles), 5 cm H_2O (triangles), and 10 cm H_2O (squares) during gas ventilation (GV), at full, and during evaporation phases E1, E2, and E3. Values are mean ± 2 SEM. There was a difference between GV and E2 and E3 ($p \leq 0.05$), but not between GV and E1. There was also a difference between PSV of 10 cm H_2O and the other 2 PSV levels ($p \leq 0.05$).

more work to expand the heavier PLV-filled lung. However, it may be a misnomer to call it a liquid-filled lung, since the perflubron is held in the periphery while PEEP is applied to the ETT, and the trachea is relatively free of perflubron. Thus, the presence of perflubron may not affect triggering.

A study by Hummler et al[16] examined the WOB associated with PLV in spontaneously breathing rabbits. WOB was compared between GV and a liquid-filled lung, using a different perfluorocarbon (Rimar 101, Miteni, Italy). They observed greater WOB in the liquid-filled lung than in the gas-filled lung. That study contrasts our findings, in which there was no difference in WOB_R, WOB_E, or WOB_T between the liquid-filled and the gas-filled lung. We found increased WOB during lung filling, but, once the lung was full, WOB returned to the level of the gas-filled lung. Study design differences (including the type of perfluorocarbon used, the animal species, the method of perfluorocarbon administration, and the length of the study) may explain these conflicting results. In our study we maintained the animals on PLV for 8–10 hours, whereas in the Hummler experiment animals were only studied for 2–3 hours.

In our study there was no evidence of increasing fatigue in the animals during PLV, as measured by the animal's effort, respiratory rate, and WOB. In addition, since T_I/T_{TOT} is a measure of endurance, if respiratory muscles fatigue, more time is spent in inspiration, thus T_I/T_{TOT} increases. Although there was a slight increase in T_I/T_{TOT} during our experiments, there was no significant change in T_I/T_{TOT} between GV and PLV. T_I/T_{TOT} alone is not an adequate measure of fatigue.

Theoretically, the mode of ventilation can affect WOB during PLV. In our study, we chose pressure-controlled ventilation rather than volume-controlled ventilation. A recent study by Kallet et al[25] found that WOB_R was significantly lower with pressure-controlled ventilation than with volume-controlled ventilation (0.59 ± 0.42 j/L vs 0.70 ± 0.58 j/L, $p \leq 0.05$). Kallet et al hypothesized that this difference was due to increased imposed WOB_R during volume-controlled ventilation. In addition, it was evident in volume-controlled ventilation that WOB_E, associated with lung inflation and chest wall displacement, increased. These differences were most significant with increased patient effort.

No previous study has examined the effect of pressure support during PLV. However, pressure support has been shown efficacious in decreasing WOB during mechanical ventilation. The effectiveness of pressure support is dependent on both patient[26] and equipment factors.[27]

Using 3 different ventilators with 9 intubated adults undergoing weaning in an intensive care unit, Mancebo et al[26] found that WOB_T was 38–64% less with the addition of PSV of 15 cm H_2O.

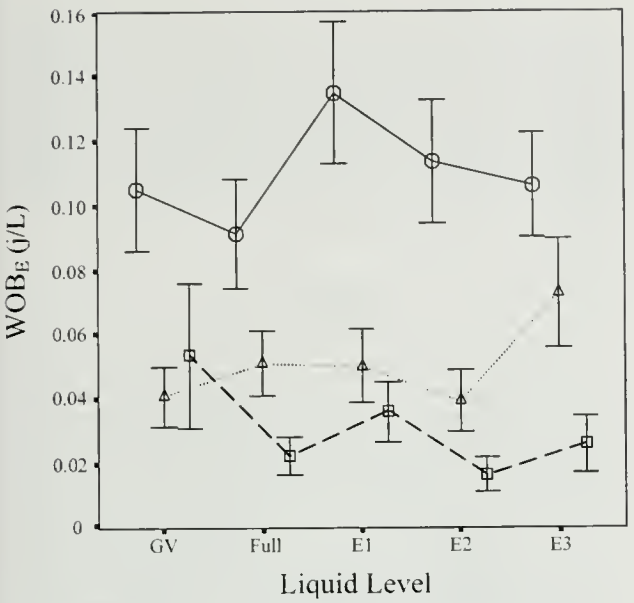

Fig. 7. Elastic work of breathing (WOB_E) at pressure-support ventilation (PSV) levels of 0 cm H_2O (circles), 5 cm H_2O (triangles), and 10 cm H_2O (squares) during gas ventilation (GV), at full, and during evaporation phases E1, E2, and E3. Values are mean ± 2 SEM. There was no difference between GV and the other liquid levels. WOB_E appeared to decrease with the addition of PSV, but the differences were not significant.

One potential limitation of our study may be our use of a healthy animal model and 5 cm H_2O PEEP, which may not have been adequate to maintain FRC in our healthy animals. A recent study by Ferreyra et al[28] found that PEEP of ≥ 10 cm H_2O was required to normalize gas delivery to FRC in a healthy animal model during PLV. Other studies have found improvements in FRC and gas exchange, without hemodynamic compromise, when PEEP > 5 cm H_2O was used during PLV.[29,30]

Although our study was arbitrarily ended at 8–10 hours, the animals did not appear to experience deleterious effects from breathing spontaneously during PLV. That finding suggests that an even longer duration of PLV would be possible. After each experiment was completed, each animal had residual perflubron removed by gravity drainage and was returned to its pen. All the animals survived.

There may be distinct advantages to allowing patients to breathe spontaneously once their lungs have been filled with perflubron. First, one of the concerns during PLV is the mobilization of secretions and the potential to plug airways, which may be reduced during spontaneous breathing PLV. Second, by allowing patients to breathe spontaneously, with work minimized but not eliminated, the duration of ventilation may be reduced. Although in clinical practice it is often desirable to minimize WOB, in me-

Table 1. Pulmonary Monitor Data*

PSV (cm H_2O)	Phase	f (breaths/min)	$\dot{V}_E$ (L/min)	ΔP_{AW} (cm H_2O)	PIP (cm H_2O)	ΔP_{es} (cm H_2O)	PTP (cm $H_2O \cdot s \cdot min^{-1}$)	$P_{0.1}$ (cm H_2O)	T_I/T_{TOT}
0	Gas ventilation	23 ± 5	2.2 ± 0.4	9.5 ± 4.4	13.3 ± 3.2	5.4 ± 4.3	49 ± 31	2.9 ± 3.2	0.24 ± 0.03
	Filling†	29 ± 7	2.5 ± 0.6	8.8 ± 1.1	12.2 ± 1.1	5.2 ± 3.4	51 ± 29	3.4 ± 2.5	0.25 ± 0.04
	Full	31 ± 8	2.4 ± 0.5	9.1 ± 2.2	12.5 ± 2.1	4.2 ± 1.4	42 ± 18	2.5 ± 0.9	0.28 ± 0.05
	Evaporative‡	35 ± 12	2.7 ± 0.8	8.2 ± 1.8	11.0 ± 1.1	4.4 ± 1.5	48 ± 26	2.9 ± 1.7	0.29 ± 0.06
5	Gas ventilation	23 ± 5	2.4 ± 0.5	10.1 ± 1.7	13.9 ± 0.6	4.1 ± 5.0	32 ± 31	2.6 ± 3.2	0.23 ± 0.05
	Filling	25 ± 6	2.4 ± 0.4	10.4 ± 1.4	14.1 ± 1.1	4.0 ± 3.3	33 ± 29	3.1 ± 2.9	0.22 ± 0.03
	Full	31 ± 7	2.5 ± 0.3	9.7 ± 0.6	13.4 ± 0.8	3.5 ± 1.2	30 ± 11	2.5 ± 1.5	0.26 ± 0.04
	Evaporative	30 ± 7	2.6 ± 0.6	10.0 ± 1.8	13.2 ± 1.1	3.5 ± 1.9	31 ± 19	2.8 ± 2.1	0.26 ± 0.06
10	Gas ventilation	17 ± 2	2.1 ± 0.2	13.7 ± 1.6	17.6 ± 0.7	3.4 ± 4.7	19 ± 26	2.7 ± 3.0	0.18 ± 0.03
	Filling	20 ± 5	2.2 ± 0.3	13.6 ± 0.9	17.3 ± 0.8	2.9 ± 2.9	18 ± 21	3.0 ± 2.7	0.19 ± 0.03
	Full	24 ± 7	2.3 ± 0.4	13.1 ± 0.6	16.9 ± 0.8	2.3 ± 1.8	12 ± 09	2.1 ± 1.9	0.22 ± 0.05
	Evaporative	24 ± 7	2.4 ± 0.6	12.7 ± 1.1	16.6 ± 0.7	2.3 ± 1.4	13 ± 10	2.2 ± 1.5	0.23 ± 0.05

*Synchronized intermittent mandatory ventilation plus pressure-controlled ventilation with pressure-support, positive end-expiratory pressure 5 cm H_2O, and flow triggering for the phases of liquid ventilation and levels of pressure support.
Values are mean ± SD
PSV = pressure support ventilation
f = respiratory rate
$\dot{V}_E$ = minute volume
ΔP_{AW} = change in airway pressure
PIP = peak inspiratory pressure
ΔP_{es} = change in esophageal pressure
PTP = pressure-time product
$P_{0.1}$ = mouth occlusion pressure 0.1 s after the onset of inspiratory effort (respiratory drive)
T_I/T_{TOT} = ratio of inspiratory time to total breathing time (respiratory time fraction)
†Average of 10 mL/kg and 20 mL/kg filling phases
‡Average of E1, E2, and E3 evaporative phases

chanically ventilated pediatric patients the clinician must balance the additional work imposed by the ventilator apparatus and the patient's disease state.

For measuring WOB, P_{es} provides an indirect measure of intrapleural pressure and thus of the distending pressure of the lung and chest wall. Another potential limitation of our study is whether P_{es} is a true representation of intrapleural pressure in an animal with a perflubron-filled lung. Intrathoracic factors can affect the measurement of P_{es}. For example, Milic-Emili and Petit[31] showed that the compression exerted by the mediastinal contents in the supine position might modify P_{es}. However, these effects are only seen in measurements of inspiratory work and are cancelled out by expiratory work changes, allowing for accurate measurement of WOB_T. Another source of error during measurement of WOB is that the calculation of WOB_E requires measuring the chest wall relaxation curve, which in our study was measured by administering a short-acting neuromuscular blocking agent. Once perflubron was administered, the weight of the substance in the lung could have distorted the chest wall, thus causing erroneous measurements of WOB_E and WOB_T. It has been shown that distortions of the passive rib cage-abdominal configuration occur at high levels of ventilation[32] or, theoretically, by increasing airway resistance or exaggerated effort in triggering the ventilator may increase elastic work. In the previously cited study,[32] Goldman et al, using separate volume-pressure diagrams of the rib cage and abdomen, showed that work due to chest wall distortion may increase WOB_T as much as 25% in exercising subjects. In our study we did not observe any evidence of increased effort during triggering or increased airway resistance. Also, there were no significant differences in WOB_E between the perflubron-filled lungs and the gas-filled lungs.

A possible limitation in our experimental setup is the potential effect of perflubron vapor creating artifacts in the pneumotachograph readings, thus affecting our WOB measurements. However, preliminary studies in our laboratory found no evidence of such an effect. In addition, the pneumotachograph was protected from perflubron contamination by a heat-and-moisture exchanger. A previous study showed that perflubron vapor does not affect the accuracy of the pneumotachograph or flow transducer in a mechanical ventilator.[33]

Conclusions

In conclusion, WOB was not different between the GV lung and the PLV lung when the perflubron level was maintained at FRC. However, since WOB was higher than GV lung at perflubron levels less than FRC, there must be caution when allowing patients to breathe spontaneously during PLV if the perflubron level is not maintained at FRC. At perflubron levels less than FRC, WOB can be decreased by PSV, so when allowing patients to breathe spontaneously during PLV at perflubron levels less than FRC the clinician may want to increase the PSV level to compensate for increased WOB. Further study of PLV is required in animals with lung injury and should be examined for extended periods before spontaneous breathing during PLV can be recommended.

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The Effects of Motion Artifact and Low Perfusion on the Performance of a New Generation of Pulse Oximeters in Volunteers Undergoing Hypoxemia

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INTRODUCTION: Motion artifact and low perfusion often lead to faulty or absent pulse oximetry readings in clinical practice. OBJECTIVE: Determine the impact of motion artifact and low perfusion on newly introduced pulse oximetry technologies during hypoxemic episodes in healthy volunteers. METHODS: Five different pulse oximeters from 4 manufacturers (the Datex Ohmeda 3900P; the Agilent; the Nellcor N-3000; the Nellcor N-395; and the Schiller OX-1, which is the European version of the Ivy SatGuard 2000 with Masimo SET) were compared with respect to their ability (separated or in combination) to provide accurate readings in the presence of motion artifact and low perfusion. Four of these oximeters represent the latest available oximetry technology, and one (the N-3000) represents a previous generation of oximeters. Oxygen saturation values (S_{pO_2}) and pulse rate from the oximeters were recorded during episodes of induced hypoxemia in 10 healthy volunteers. Standardized and repeatable motion artifacts were generated by a motion machine and by having the test subject perform tapping and scratching motions. Perfusion to the finger was reduced by an inflatable balloon impinging on the brachial artery. The pulse oximetry readings from the test oximeters were compared to readings from control pulse oximeters on the unperturbed reference hand. The pulse rates from the test oximeters were compared to the electrocardiographically-measured heart rate. RESULTS: The frequency of faulty readings was increased by increasing motion interference and decreasing perfusion. The S_{pO_2} deviation was within ± 3% of the reference reading > 95% of the time for all instruments during the control desaturation period in the absence of motion and with normal perfusion. With the combination of motion and low perfusion, the S_{pO_2} error was within ± 3% less than 62% of the time for all oximeters tested. A significant difference in the frequency of large S_{pO_2} errors was observed only in the direct comparison of the N-395 and N-3000. The N-395 exhibited less frequent S_{pO_2} error exceeding 6% of S_{pO_2} in the combination of the most challenging situations (motion and motion with reduced perfusion). In the same situation the Datex-Ohmeda 3900P and Nellcor N-3000 showed significantly higher pulse rate errors than the other devices (Datex-Ohmeda 3900P 53% of the time and N-3000 37% of the time). CONCLUSIONS: The established model of creating motion artifact and low perfusion is capable of simulating a hierarchy of severe clinical situations. With solely motion or solely reduced perfusion the percentage of errors exceeding ± 3% of S_{pO_2} increased by 20% and 10%, respectively, compared to the control period. Simultaneous presence of motion and reduced perfusion leads to a relative incidence of > 35% of errors > 3% of S_{pO_2} for the various oximeters. In this situation the N-3000 and the Datex-Ohmeda 3900P exhibited differences between estimated pulse rate and electrocardiographically-measured heart rate > 25 beats/min > 37% of the time.

Key words: pulse oximetry, artifact simulation, motion, reduced perfusion, desaturation, monitoring.

[Respir Care 2002;47(1):48–60]

Introduction

Pulse oximetry is part of routine basic monitoring in anesthesia, along with electrocardiographic (ECG) and blood pressure monitoring. Pulse oximetry is commonly employed in nearly all areas where patients are at risk of hypoxemia.

In clinical practice, pulse oximetry has to cope with many factors that influence S_{pO_2} accuracy. In a randomized study of pulse oximetry performance,[1] the pulse oximetry failure rate (temporarily and completely abandoned pulse oximetry monitoring) was 2.5% of all monitored patients, and it increased to 7.2% in patients with American Society of Anesthesiology status 4. The main reasons (59%) for temporary and complete pulse oximetry failure in that study were *restricted peripheral perfusion* and *movement artifacts* due to patient restlessness. Minor reasons (41%) for pulse oximetry failure were technical or practical problems (not further defined) and unknown.

In a retrospective study by Reich et al, the incidence of intraoperative loss of pulse oximetry readings for > 10 minutes was 9.2%.[2] Predictors of pulse oximetry failure included American Society of Anesthesiology status 3, 4, and 5 patients undergoing vascular or cardiac surgery, and restricted finger perfusion caused by hypothermia or hypotension.

The conditions that are most challenging for pulse oximeters are patient motion and low perfusion, which can be present in critical situations such as surgery, during patient transport, in emergency departments, and in neonatal intensive care units. We believe that a clinically relevant performance test of pulse oximeters should include a standardized simulation of motion and restricted perfusion during changes in arterial oxygen saturation. So under those conditions we tested instruments from the most recently introduced generation of pulse oximeters from 4 manufacturers. Our goal was to address the question, "Do the new oximeters improve the accuracy of tracking S_{pO_2} and pulse rate during induced patient hypoxemia in the presence of motion artifact and low perfusion?"

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The new-generation instruments we tested were: the 3900P, software version 2.000/04.000 (Datex Ohmeda, Helsinki, Finland); the Agilent with CMS monitor software Rev B.0 (Philips Medical Systems, Böblingen, Germany); Nellcor N-395 with OxiSmart XL (software version 1.6.1.0) (Tyco Healthcare Group, Pleasanton, California); the Ivy SatGuard 2000 with Masimo Signal Extraction Technology (SET) (Model 2000, Ivy 2356–00-04 REV 01 DSP 2.2.0.0, MS1:HW REV: A ID:2 SMC: 1.0.2.2, sold by the Swiss company Schiller as model OX-1) (Ivy Biomedical Systems, Branford, Connecticut; Masimo, Irvine, California). The Nellcor N-3000 (software version 3.03 001) was also tested as a representative of the previous generation of oximeters.[1]

Methods

Ten healthy volunteers (8 men and 2 women, 30–37 years of age) participated in the study. The study was approved by the Ethics Committee of the Medical University of Lübeck. Prior to the study, participants were checked by an independent cardiologist and had no evidence of vascular, cardiopulmonary, or other systemic disease. After written informed consent was obtained, all volunteers participated in a training desaturation procedure one month before the motion study, to get used to the test situation.

To assess the effect of motion artifact and low perfusion on the pulse oximeters' ability to accurately detect changes in arterial oxygen saturation, we tested the oximeters during a controlled desaturation procedure in which both hands were at heart level. The reference oxygen saturation was recorded on the right hand (reference hand), using 2 Nellcor N-3000 pulse oximeters. The left hand (test hand) was subjected to motion and low perfusion. Heart rate was recorded with an ECG, using electrodes placed on the chest.

Controlled Desaturation Procedure

Participants breathed through a tight-fitting face mask connected to a Trajan 808 (Dräger Medizintechnik, Lübeck, Germany) with fresh gas supply of 14–17 L/min, using a valveless high-flow system. The expiration side of the Y-piece was 1 m in length, providing uncontrolled inspiratory gas mixture. Fraction of inspired oxygen was varied by adjusting oxygen and nitrogen with the system's flow meters. Inspiratory and expiratory oxygen and carbon dioxide concentration and breathing pattern were continuously monitored by a Datex Ultima V (Datex Medical Instruments, Helsinki, Finland). The bidirectional, pressure-based flow sensor (D-lite, Datex Medical Instruments, Helsinki, Finland) with side stream system was introduced into the air flow between the Y-piece and the mask.

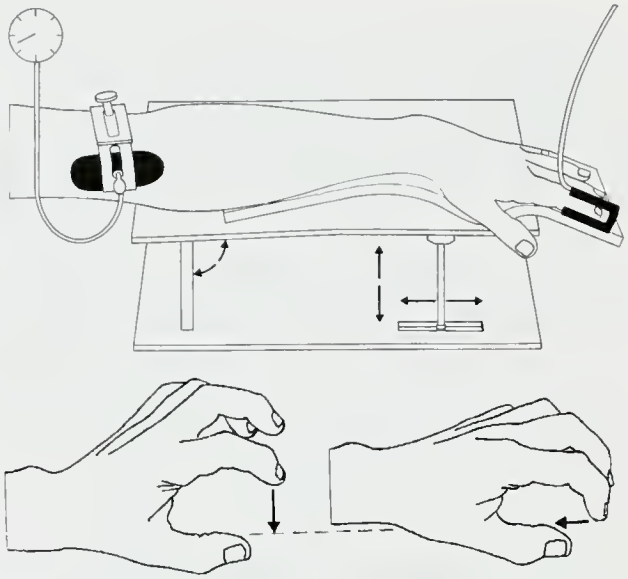

Fig. 1. The motion machine (top) and the tapping (bottom left) and scratching (bottom right) motions. The left part of the table supporting the arm and hand is pivoted and the rod on the right side moves the table up and down. The arm pressure cuff for the reduction of perfusion is indicated at the left side.

Motion

We used a mechanical motion generator to move the test hand with repeatable computer-generated irregular patterns, to produce a standardized motion artifact (Figure 1). The maximum motion amplitude was 38 mm, which is a range used in previous studies and which represents a substantial challenge to pulse oximeters.[3] The minimum period for a 38 mm movement was about 0.1 second. Irregular patterns were thought to be more realistic than simple repetitive sine wave motions. A similar motion simulator was used by Barker and Shah in a previous study.[3] Additionally, in order to simulate more clinically relevant motion patterns, we asked each volunteer to perform a tapping motion for 120 seconds, followed 60 seconds later by a scratching motion for 120 seconds. A training video showing the desired motion patterns was presented to the volunteers before the study. The voluntary motion patterns were synchronized using a nonperiodic acoustic signal provided to the volunteers to define start and stop points for each tapping and scratching motion.

Low Perfusion

We produced varying levels of finger perfusion by compressing the brachial artery of the left upper arm. To control arterial perfusion we used a specially designed clamp that encircles the arm, without causing venous congestion, while holding a balloon in a fixed position above the artery. A similar system was established in a previous study.[4] The perfusion was controlled by inflating the balloon under continuous pressure measurement. The balloon pressure was increased until the modulation depth of the photoplethysmographic signal was decreased to less than 2%. The perfusion on the reference hand was monitored with a Nellcor N-395 pulse oximeter. In this way the perfusion of the fingers was held at a nearly constant level, lower than the perfusion of the reference hand.

Three of the test instruments provide a pulse strength index that is related to the perfusion or percent modulation of the photoplethysmographic signal. In the case of the Agilent monitor it is called *perfusion index* and is the percentage modulation of the transmission signal corrected for the influence of saturation. Normal values are between 0.1% and 20%. The Nellcor N-395 provides a pulse amplitude indicator, which is a number between 0 and 200. The Datex Ohmeda 3900P provides a pulsation index ranging from 0.00 to 9.99. For all instruments, a higher value means larger amplitude pulses. To make the values comparable, the Nellcor pulse amplitude value was divided by 10 and the Datex-Ohmeda pulsation index was multiplied by 2. All values are thus comparable to the Agilent percentage value, ranging from 0 to 20%. This variable was used to monitor our method of reducing perfusion.

Protocol

Figure 2 describes the procedure. The subject's oxygen saturation started at 100% and was decreased to 90%, where it was held constant for about 1 minute. Then it was decreased further to about 80%, where it again was held constant. This was followed by an increase to 90% (constant period again), and then an increase to 100% oxygen saturation, where it was held constant for at least 2 minutes. Motion started at the 100% plateau. In Figure 2, motion periods and periods when the balloon was inflated are indicated by bars. The maximum pressure was adjusted individually for each volunteer. At the end of the study, we added another desaturation period, which involved low perfusion but no motion artifact.

Data Acquisition

In order to report the displayed oxygen saturation and the alarm status of the instruments, we continuously recorded the following variables:

- S_{pO_2} of every instrument simultaneously (control pulse oximeters and pulse oximeters being tested)
- Pulse rate
- ECG-measured heart rate, recorded with the Agilent CMS monitor

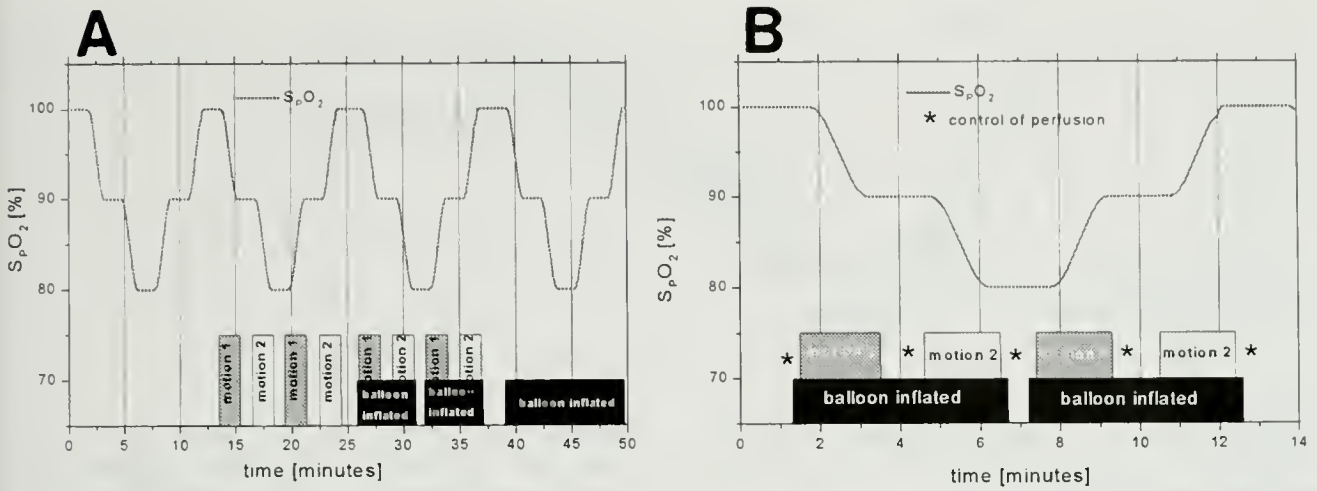

Fig. 2. Timing of the study protocol. A: Overall timing. B: Enlargement of the third desaturation period. The curve represents the oxygen saturation measured via pulse oximetry (S_{pO_2}). The shaded and clear bars indicate the times when motion was present. The black bars represent the times when the balloon was inflated. The asterisks mark the times when the reduction of the pulse amplitude, caused by the balloon inflation, was controlled.

- An index related to the perfusion or percent modulation (Agilent perfusion index, Nellcor N-395 pulse amplitude, Datex-Ohmeda pulsation index)
- All alarm variables and warnings

Settings

The alarm limit for low saturation was set to 92% for all instruments. The pulse limits were set to 40 (low pulse rate) and 150 (high pulse rate).

The Agilent response time was set to medium, which is about 10 seconds.

The Datex-Ohmeda averaging time was set to 6 seconds.

The Ivy 2000 with Masimo SET was set to 10 seconds averaging, and its sensitivity was set to normal.

The response time of the Nellcor pulse oximeters is not accessible to users. It is nominally about 7 seconds.

Sensors

For the sensors on the test hand we used adhesive sensors so as to avoid the additional contribution to motion artifact that can be expected from the more massive reusable sensors. The sensors we used were:

- OxyTip adult sensor (Datex-Ohmeda)
- Nellcor Oxisensor II D25 (Tyco Healthcare Group) for Agilent, Nellcor N-3000, and Nellcor N-395
- Masimo SET LNOP Adt (Masimo) for the Ivy 2000 with Masimo SET

All sensors were protected with opaque covers to prevent light from one sensor reaching another sensor and interfering with performance. The pulse oximeters on the test hand were positioned on randomly-selected fingers, differently for each volunteer.

Reference

Two Nellcor N-3000 pulse oximeters equipped with DS-100A Durasensors on fingers of the nonmoving and normally perfused right hand represented the reference. These 2 reference oximeters were used to control the desaturation procedure and served as the standard for comparing the pulse oximetry readings from the moving test hand. The mean values of the pulse oximetry readings from those 2 devices had been compared against arterial blood samples in previous desaturation procedures, and the mean value was an accurate measure of the arterial oxygen saturation.[5]

Statistics

The continuously recorded data were analyzed with respect to the number of deviations of pulse oximetry readings and pulse rate, and with respect to alarms. The analysis was divided among various periods, each period representing a different level of signal interference:

Period 1: no motion, normal perfusion
Period 2: no motion, low perfusion
Period 3: motion, normal perfusion
Period 4: motion, low perfusion

In periods 3 and 4, all motion patterns (machine motion, tapping, and scratching) were merged.

Pulse oximetry error was defined as the S_{pO_2} of the test hand minus the S_{pO_2} of the reference hand. The S_{pO_2} of the test hand was the reading of the pulse oximeter under test

Table 1. Definition of Error Categories*

False Cumulative	Category	False Low	Subcategory	False High	Subcategory
$\lvert\Delta S_{pO_2}\rvert \leq 3$	1	$-3 \leq \Delta S_{pO_2} \leq 0$	1_L	$0 \leq \Delta S_{pO_2} \leq 3$	1_H
$\lvert\Delta S_{pO_2}\rvert \leq 6$	2	$-6 \leq \Delta S_{pO_2} < -3$	2_L	$3 < \Delta S_{pO_2} \leq 6$	2_H
$\lvert\Delta S_{pO_2}\rvert \leq 10$	3	$-10 \leq \Delta S_{pO_2} < -6$	3_L	$6 < \Delta S_{pO_2} \leq 10$	3_H
$\lvert\Delta S_{pO_2}\rvert > 10$	4	$\Delta S_{pO_2} < -10$	4_L	$\Delta S_{pO_2} > 10$	4_H

*The first two columns define the absolute error categories. In the other columns these categories are subdivided into false low (index L) and false high (index H) S_{pO_2} values.
$\lvert\Delta S_{pO_2}\rvert$ = absolute value of the error of the oxygen saturation value measured via pulse oximetry.
S_{pO_2} = oxygen saturation value measured via pulse oximetry.
See text for explanation of subcategories.

conditions. The S_{pO_2} of the reference hand was the mean value of the S_{pO_2} readings of the 2 Nellcor N-3000s on the reference hand.

For the S_{pO_2} error, categories were defined as shown in Table 1. The neutral range was defined as ± 3% of the S_{pO_2} value, since that is the range that many pulse oximetry manufacturers define as the range of accuracy in the presence of motion (without motion it is often given as ± 2% of the S_{pO_2} value). These categories are subdivided into false low (index L) and false high (index H) S_{pO_2} readings.

From other studies it is known that the incidence of large positive or negative pulse oximetry errors (ΔS_{pO_2}) increases as an effect of poor signal quality, as occurs during motion and low perfusion. A broadening of the error distribution is observed, whereas the mean value of ΔS_{pO_2} is less affected. For that reason, when specifying the accuracy of their instruments, most manufacturers provide larger standard deviations under motion conditions. Comparing pulse oximeters in different situations requires a test that is sensitive to the error distribution, so we applied the Wilcoxon matched-pairs signed-rank test to the absolute values of the errors ($\lvert\Delta S_{pO_2}\rvert$). In addition, to test the incidence of S_{pO_2} errors exceeding 6% and 10%, respectively, a sign test was used. For this purpose new variables Y_{PO1} and Y_{PO2} were generated:

$$Y_{POi} = \begin{cases} 1 & if \quad \Delta SpO_2 > c \\ 0 & else \end{cases} \qquad (1)$$

for c = 6% S_{pO_2} and c = 10% S_{pO_2}.

The whole protocol described above was performed for each volunteer. S_{pO_2} readings from the pulse oximeters

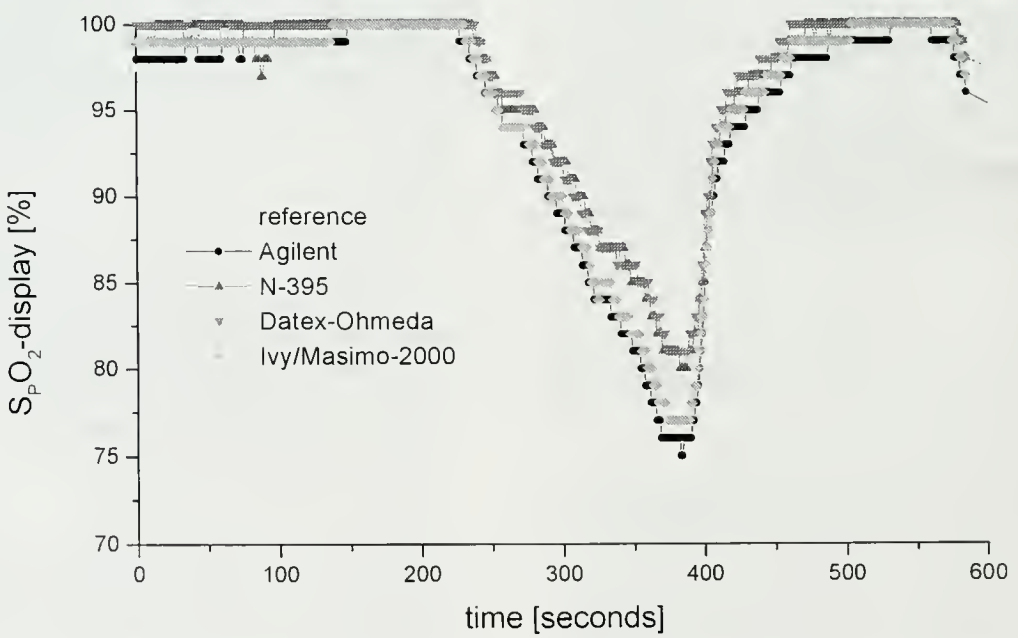

Fig. 3. Oxygen saturation measured via pulse oximetry (S_{pO_2}) versus time for 4 test oximeters and the reference S_{pO_2} during a desaturation in Period 1 (no motion and normal perfusion). The S_{pO_2} differences between the oximeters are within the defined range of ± 3%.

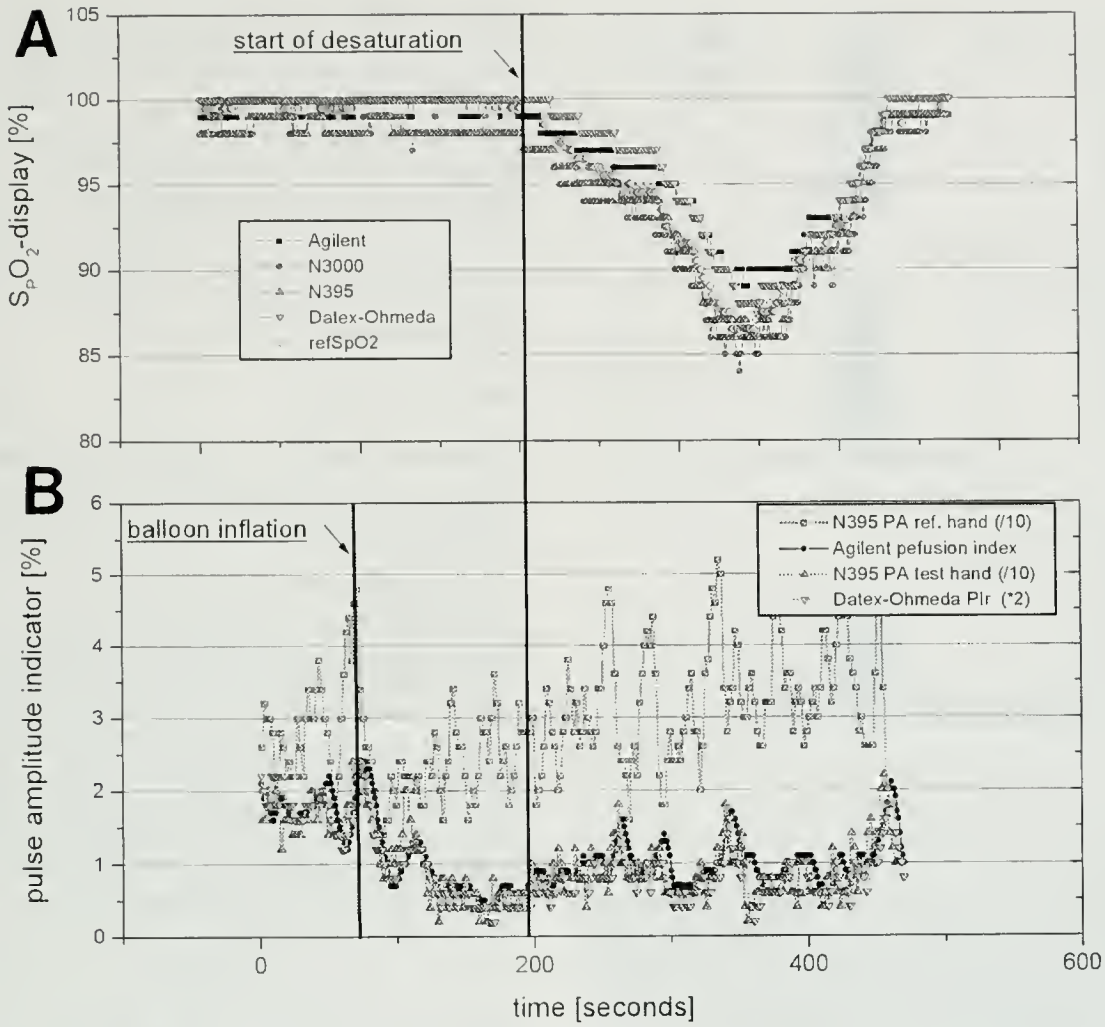

Fig. 4. A: Oxygen saturation measured via pulse oximetry (S_{pO_2}) versus time during a desaturation in Period 2 (low perfusion, no motion). B: Pulse amplitude (PA) indicator versus time during a desaturation in Period 2 (low perfusion, no motion). The perfusion of the test hand is reduced after balloon inflation, while the perfusion of the reference hand (pulse amplitude indicator of Nellcor N-395, PA ref hand) remains constant.

were sampled at 1 Hz. However, pulse oximeters do not create new values every second, so adjacent data points are not independent, because of the different averaging algorithms employed in the various oximeters. In order to avoid an unreasonable level of data generation, data were resampled at 0.1 Hz. This new data set was treated as independent data used to generate the error distribution, which served as the basis for the statistical tests (in Figs. 3 and 4 the original 1 Hz sampling rate is displayed). These tests were applied separately for each volunteer, since it is known that pulse oximeters show subject-specific performance because of interindividual variability.

For every pair of pulse oximeters, the S_{pO_2} errors of those 2 instruments were compared directly at the selected time points. With 5 pulse oximeters there are 10 possible pairs. As only 4 pulse oximeters were attached to the hand of each volunteer (we did not use the thumb), for each pair of instruments the data of 6 volunteers were available.

Let us now consider one particular period and one pair of pulse oximeters. In order to account for possibly subject-dependent variances, we applied 2 tests (Wilcoxon signed-rank test and sign test), to the data of each volunteer separately. This resulted in Z values Z_1 through Z_6 (ie, transformed p values, one for each volunteer). A combined Z value for the 6 volunteers was computed using the formula:

$$Z_{sum} = \Sigma Z_i / \sqrt{n} \qquad (2)$$

Under the null hypothesis of no difference between the 2 instruments, every single Z_i as well as Z has a standard

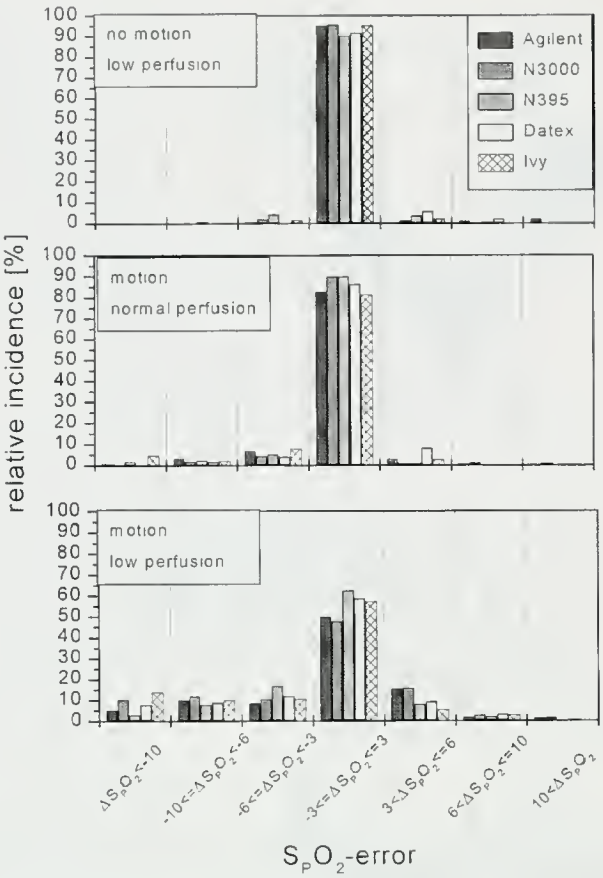

Fig. 5. Relative incidence of cases within the error categories (4L to 4H) for Periods 2 (top), 3 (middle), and 4 (bottom). The increasing challenge for the pulse oximeters is visible in the increase of column heights for the higher error categories. In the period with motion and low perfusion, all instruments tested showed high S_{pO_2} (oxygen saturation measured via pulse oximetry) errors.

normal distribution. This leads to a p value of p = NCDF (Z), where NCDF denotes the standard normal distribution function.

Note that we have 40 different p values corresponding to all combinations of 4 periods and 10 pairs of pulse oximeters. In order to account for multiple testing, we made a Bonferroni adjustment and replaced each individual p value with the value of p × 40. Then any p value of ≤ 0.05 is significant at that level and can be interpreted.

The times when some pulse oximeters displayed a zero reading or no reading are listed separately.

Pulse rate error was defined as the pulse rate measured by pulse oximetry minus the heart rate measured via ECG and recorded using an Agilent CMS monitor. For the pulse rate error, 2 error categories were defined: pulse error ≤ 10 beats/min and pulse error ≤ 25 beats/min. We defined the 10 beats/min range as the neutral range. This represents the range covering more than 90% of all readings when no motion is present. The relatively large deviations between oximetry-measured pulse rate and ECG-measured heart rate are caused in part by the differing averaging times of the pulse oximeters and the ECG instrument. The pulse oximeters use the averaging times described above (approximately 10 s), whereas the ECG monitor computed a beat-to-beat heart rate or computed heart rate using 2 neighboring RR intervals. For that reason, the natural heart rate variability was the cause of at least some of the discrepancy between the 2 measurements. The statistical analysis was performed in the same way as described for the S_{pO_2} error.

Selection of Periods

The motion period was defined as the period in which the hand was moving, plus the 20 seconds after the end of the motion, to take into account the pulse oximeters' response times.

Despite setting the oximeters' response times to similar values, there were small differences in dynamic behavior between the various pulse oximeters. We therefore only used periods in which the changes in the S_{pO_2} values of the reference pulse oximeters were < 0.3% of S_{pO_2} per second. This was done by forming the first derivative of the S_{pO_2} signal and neglecting all periods wherein the $|\Delta S_{pO_2}/s|$ was < 0.3, and, in addition, the periods 5 seconds before and after these intervals. That included all periods with desaturation and resaturation, with maximum S_{pO_2} alterations of 20% of S_{pO_2} per minute.

Results

Figure 3 shows an S_{pO_2} recording during a desaturation in Period 1 (no motion and normal perfusion). The differences between the oximeters are within the defined range of ± 3%.

Change of Perfusion

Figure 4B shows the change of perfusion caused by inflating the balloon positioned at the brachial artery of the test arm, while figure 4A demonstrates the corresponding desaturation values of the pulse oximeter during this period. The SpO2 data from the various pulse oximeters of the test hand and reference hand are in a good agreement.

S_{pO_2} Error

Figure 5 shows the percentage of the total time the S_{pO_2} error was in categories 4L (low) through 4H (high) during Periods 2–4. The increasing challenge for the pulse oximeters is visible in the increase of column heights for the higher error categories. In the period with motion and low

Table 2. Results of Wilcoxon Matched-Pairs Signed-Rank Test on the Absolute Errors of the S_{pO_2} Values and the Results of Sign Test

Pair	Wilcoxon Test				Sign Test		
					$\lvert\Delta S_{pO_2}\rvert > 6$		$\lvert\Delta S_{pO_2}\rvert > 10$
	Period 1	Period 2	Period 3	Period 4	Period 1–3	Period 4	Period 1–4
Datex–Agilent	NS	NS	NS	NS	NS	NS	NS
Datex–N-395	NS	NS	NS	NS	NS	NS	NS
Datex–N-3000	Datex*	NS	NS	NS	NS	NS	NS
Ivy–Datex	NS	NS	Ivy*	NS	NS	NS	NS
Ivy–Agilent	Ivy*	NS	NS	NS	NS	NS	NS
Ivy–N-395	N-395*	NS	NS	NS	NS	NS	NS
Ivy–N-3000	Ivy*	NS	Ivy*	NS	NS	NS	NS
N-395–Agilent	N-395*	NS	NS	NS	NS	NS	NS
N-395–N-3000	N-395*	NS	NS	NS	NS	NS	NS
N-3000–Agilent	Agilent*	NS	Agilent*	NS	NS	NS	NS

$\lvert\Delta S_{pO_2}\rvert$ = absolute value of the error of the oxygen saturation value measured via pulse oximetry.
NS = no significant difference.
* = significant difference ($p = 0.05$). The more error-prone pulse oximeter is named.

perfusion, all instruments tested showed high S_{pO_2} errors. The selected Periods 2 (low perfusion) through 4 (motion and low perfusion) fulfill the requirement of representing different levels of interference.

The false low readings are more pronounced than the false high readings because the magnitude of false high readings is limited by the maximum possible S_{pO_2} value of 100%. As the true S_{pO_2} values were always in the range of 70–100%, the maximum positive deviation was 30% at the lowest saturations tested, and less for larger saturations. For false negative values there is a wider range of possibilities.

Table 2 shows the results of Wilcoxon matched-pair signed-rank test and sign test applied to the absolute values of ΔS_{pO_2}.

Figure 6 plots the means of the absolute values of ΔS_{pO_2} for the different volunteers, for the different pulse oximetry pairs, and for the different periods to show the variability among the volunteers. PO1 and PO2 denote the first and second oximeters in the direct comparison.

Although there were significant differences between the oximeters within volunteers, the net effect for all volunteers was almost always not significant by Wilcoxon signed-rank test or sign test of the error categories $> 6\%$ and $> 10\%$. Within Periods 1 and 2 there are some distributions that are significant at $p = 0.05$, but the errors in those periods are within 3% of the ΔS_{pO_2} and the differences can be explained by slightly differing calibration curves.

In Periods 3 and 4 some of the pulse oximeters failed to give any readings at all (ie, they displayed zero values). In Period 3 this occurred 2.7% of the time with the Agilent and 1.7% of the time with the Ivy 2000 with Masimo SET. In Period 4 it occurred 8% of the time with the Agilent and 0.7% of the time with the Ivy 2000 with Masimo SET.

Pulse Error

The oximeter pulse rates were compared to the ECG-measured heart rate. In the no-interference period the error distribution is Gaussian, with a width of 7% for all instruments (Fig. 7A). In the presence of motion and low perfusion the distribution is obviously widened (Figure 7B). Figure 7C shows the cumulative percentages for 2 error categories: pulse error $\leq 10\%$ and pulse error $\leq 25\%$. Table 3 shows the results of Wilcoxon matched-pairs signed-rank test and sign test applied to the absolute values of pulse errors for Periods 3 and 4. In Periods 1 and 2 no significant differences were observed. Wilcoxon and sign tests of the different error categories indicate that the Datex-Ohmeda device exhibits the worst pulse indication performance, followed by the Nellcor N-3000. There were no significant differences between the Agilent, Ivy-2000 with Masimo SET, and Nellcor N-395.

Discussion

Pulse oximeters have proven reliable for detecting the threat of hypoxemia and, together with capnography, they represent the fundamental monitoring for adverse anesthesia-related events.[6] The increasing challenges posed by clinical and ambulatory use of pulse oximeters have been met by their manufacturers; new algorithms for signal evaluation have been implemented into the devices on a regular basis. Our goal was to evaluate the performance of a new generation of pulse oximeters under conditions where interference of signal registration might be expected: move-

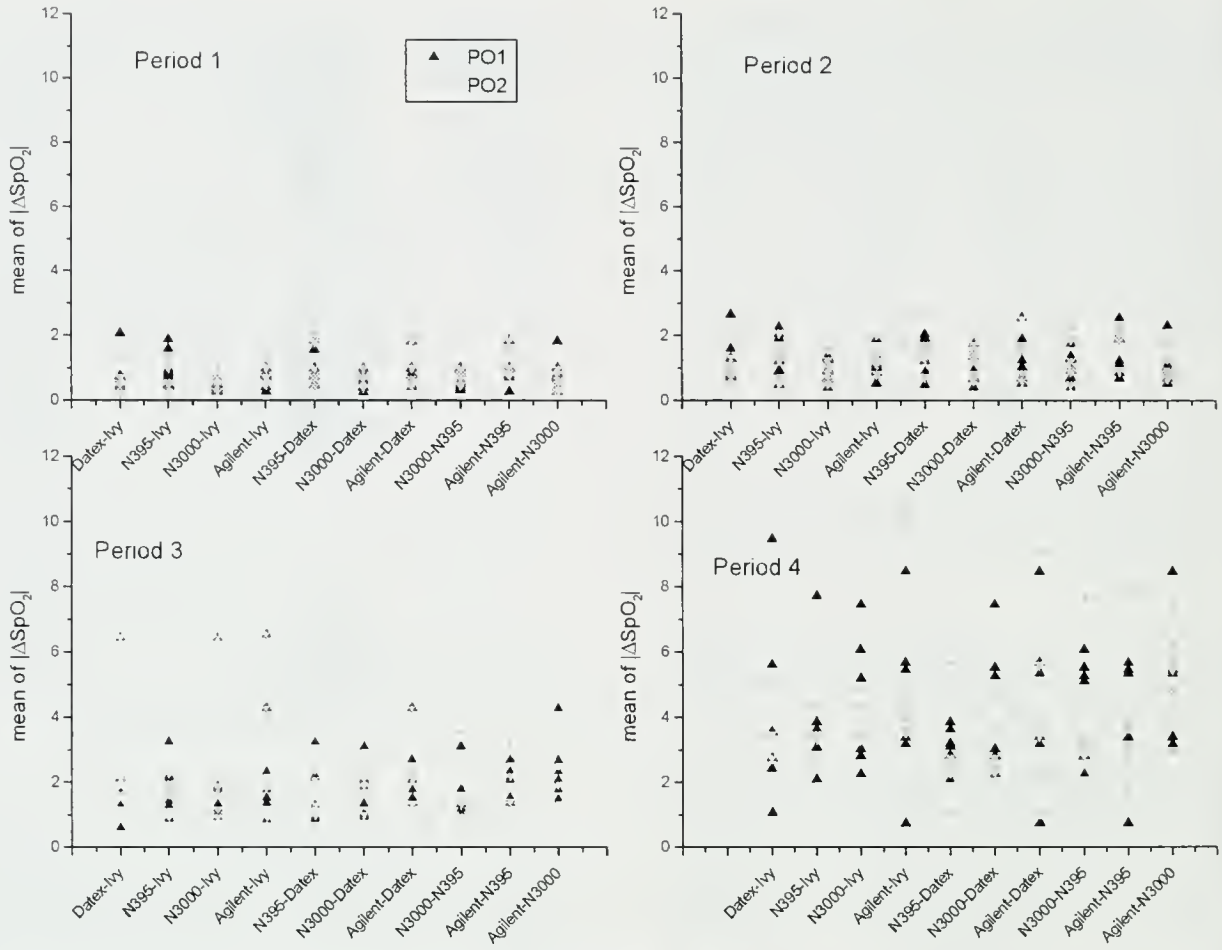

Fig. 6. Mean of the differences of the absolute values of oxygen saturation measured via pulse oximetry (ΔS_{pO_2}) in the 4 periods. There is one data point for each volunteer: the dark triangles are the data points for the first pulse oximeter (PO1) in the comparison; the light gray triangles correspond to the second pulse oximeter (PO2).

ment of the sensor-carrying finger and decreased perfusion in the measurement area. The arm motion was standardized, reproducible, and designed to produce challenging conditions for the pulse oximeters, and the motion was applied during controlled desaturation procedures in healthy volunteers, because detection of hypoxemia is the important task for pulse oximeters monitoring in the clinic. In developing this method, similarity to clinical conditions had to be guaranteed. Patients in the recovery room are often restless, and perfusion in the finger can be restricted by vasoconstriction due to peripheral hypothermia, hypovolemia, or catecholamine release.[1,2] Similar situations occur during weaning from mechanical ventilation in intensive care or during sedation for bronchoscopy.

We used motion created by a motion machine, as has been done by Barker et al,[3,7] which has the advantage of standardized reproducibility for each volunteer. For an improved modeling of clinical reality, we also introduced tapping and scratching movements, which are more challenging for the pulse oximetry sensors. We also tested oximeter performance under low perfusion conditions. Instead of using a software simulator,[8] we chose to change the arterial perfusion, and we created a control mechanism for arterial perfusion. The pulse oximeters tested indicated a change in the pulse amplitude, whereas the S_{pO_2} reading was still working properly. Unlike the procedure used by Falconer and Robinson,[4] we used not a rigid clamp but an inflatable balloon, to avoid tissue damage. All volunteers found the procedure to be agreeable and painless.

S_{pO_2} Error

For the pulse oximeters tested in the present study, the S_{pO_2} error was within ± 3% of the reference reading > 90% of the time during low perfusion and > 80% during motion (see Fig. 5). During the combination of motion and reduced perfusion the S_{pO_2} error was within ± 3% less than 62% of the time for all the tested pulse oximeters. In the present study no dramatic differences between the oximeters were observed. Although there were significant

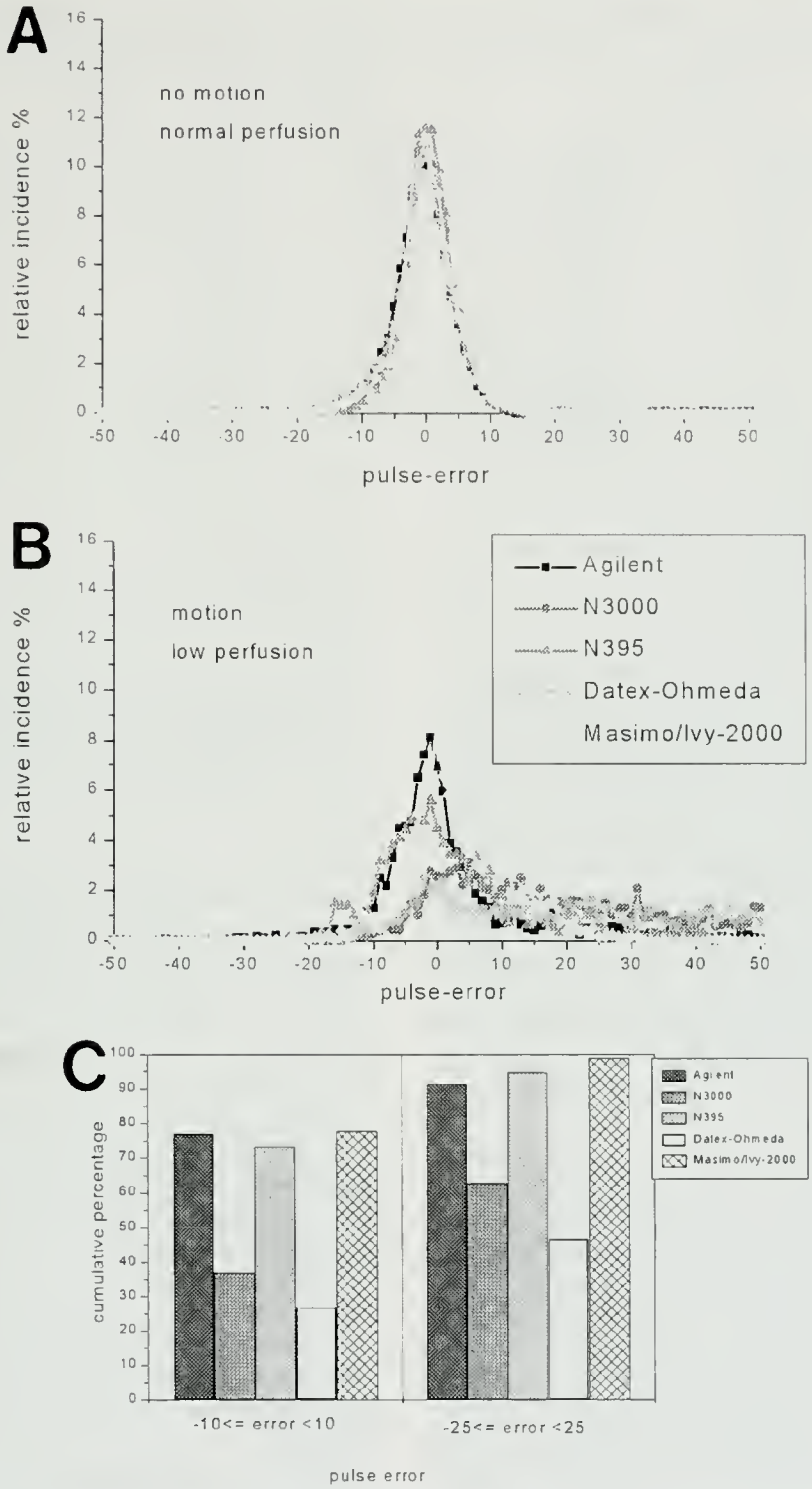

Fig. 7. Comparison of oximeter-measured pulse rate to electrocardiographically measured heart rate. Relative incidence of pulse errors (in beats/min) in (A) Period 1 (no motion and normal perfusion) and (B) Period 4 (motion and low perfusion). C: Percentage of readings within the limits pulse error $\leq$ 10 beats/min and pulse error $\leq$ 25 beats/min in Period 4 (motion and low perfusion).

Table 3. Results of Wilcoxon Matched-Pairs Signed-Rank Test on the Absolute Pulse Errors and the Results of Sign Test

Pair	Wilcoxon Test		Sign Test			
			|pulse error| > 10		|pulse error| > 25	
	Period 3	Period 4	Period 3	Period 4	Period 3	Period 4
Datex–Agilent	Datex*	Datex*	Datex*	Datex*	Datex*	Datex*
Datex–N-395	Datex*	Datex*	Datex*	Datex*	Datex*	Datex*
Datex–N-3000	Datex*	Datex*	Datex*	NS	Datex*	NS
Ivy–Datex	Datex*	Datex*	Datex*	Datex*	Datex*	Datex*
Ivy–Agilent	NS	NS	NS	NS	NS	NS
Ivy–N-395	NS	NS	NS	NS	NS	NS
Ivy–N-3000	NS	N-3000	NS	N-3000	NS	N-3000
N-395–Agilent	NS	NS	NS	NS	NS	NS
N-395–N-3000	NS	N-3000*	NS	N-3000	NS	N-3000
N-3000–Agilent	NS	N-3000*	NS	N-3000	NS	N-3000

|pulse error| = absolute value of the pulse error
NS = no significant difference.
* = significant difference ($p = 0.05$). The more error-prone pulse oximeter is named.
In periods 1 and 2 all comparisons were NS.

differences between oximeters within certain volunteers, those differences cancelled out when all the results were combined. The differences observed in Period 1 were statistically significant, but were not meaningful, because the S_{pO_2} errors remained in the neutral range in most cases ($|\Delta S_{pO_2}| \leq 3\%$). The pulse oximeters showed different responses to increasing motion levels. Some display zero readings when they failed to compute a saturation, whereas some others (eg, the Nellcor N-3000) freeze the S_{pO_2} display and warn the user with a flashing display. In this study the displayed value, although it might be frozen, was included in the analysis. The percentage of zero values was largest for the Agilent device, followed by the Ivy 2000 with Masimo SET. The other instruments did not display zero values.

A pulse oximeter's response time is the period required for the device's algorithm to orient itself reliably to alterations in oxygen saturation. To obtain a similar response from all oximeters tested, we set the response times to almost identical values (within ± 2 s). Figure 3 shows the response to a desaturation period. To impose identical requirements of the oximeters, we excluded periods with S_{pO_2} alterations exceeding 20% per minute. This lies within the required limits for rapid desaturation periods, as described by Barker and Shah.[3] Because of their non-zero response times, pulse oximeters tend to display artifact-influenced values some seconds after the artifact has stopped. We therefore included the 20 seconds after the end of the artifact into the artifact period. During profound artifact conditions, when there is no reliable signal detection, pulse oximeters tend to "freeze" the last apparently reliable value. This process leads to false readings, especially in cases where hypoxemia may be threatening.[9] The falsifying effect is strongest when a rapid change in oxygen saturation is provoked during the disrupted period. In this study we observed this freezing effect, and the S_{pO_2} errors created by those periods are included in the results.

Pulse Error

Measurement of pulse rate and its representation in visual and acoustic form is essential for supervising patients intraoperatively during anesthesia or in a pre-clinical situation of an emergency case. It has not yet been established from which source the signal should ideally be taken.[6] During anesthesia it is clinical practice to use the pulse rate display in the following way, because:

1. It underlines the credibility of the S_{pO_2} measurements. When the ECG-measured heart rate agrees with the pulse rate of the pulse oximetry it is concluded clinically that the pulse curve produced by the pulse oximeter corresponds to the patient's true pulse.

2. It can replace supervision of cardiac function when an ECG is unavailable or disrupted.

3. From the acoustic variation of the peeping tone dependent on changes in oxygen saturation, the clinician gains additional information on the oxygenation state of the patient.

Figures 7A and 7B show the pulse rate error distributions for Periods 1 and 4, respectively. Consistent with the pulse oximeters' evaluation algorithms, minor differences between ECG-measured heart rate and oximeter-measured pulse rate are normal, as can be seen from the Gaussian distribution curves in Figure 7A. The distributions of all the tested pulse oximeters are almost identical, and the differences are not statistically significant. Figure 7C shows

the cumulative percentages in the 2 error categories (pulse error $\leq$ 10% and pulse error $\leq$ 25%) for Period 4. With the Agilent, Ivy 2000 with Masimo SET, and Nellcor N-395 the pulse error was $>$ 25 beats/min $<$ 10% of the time. With the N-3000 the pulse error was $>$ 25 beats/min 37% of the time. With the Datex-Ohmeda the pulse error was $>$ 25 beats/min 53% of the time. The paired comparison of the oximeters gives a significantly higher pulse error for the Datex-Ohmeda device than all other devices in all error categories (see Table 3). The Nellcor N-3000 showed significantly higher pulse errors than the Agilent, Ivy 2000 with Masimo SET, or Nellcor N-395. There are no significant differences between the Agilent, Ivy 2000 with Masimo SET, and Nellcor N-395 in any of the error categories.

The simulated interference of Period 4 clearly represents a challenge for pulse oximetry. From a clinical viewpoint, potentially dangerous situations can arise. With pediatric patients in particular, the gap is small between adequate oxygenation and hypoxemia with the threat of bradycardia, in which case reliable detection of heart rate and S_{pO_2} are of great value.[9]

Previous studies reported better pulse oximetry performance with the Masimo signal extraction technology.[3,7,9] In high-noise settings such as in neonatal intensive care units[10] and moving patients in the recovery room,[11] a lower alarm frequency was reported. Furthermore, improved detection of hypoxemia and bradycardia was also observed.[9]

Our results herein confirm that the range of performance in pulse rate accuracy with the Ivy 2000 with Masimo SET is also achieved by the Nellcor N-395 and the Agilent CMS monitor.

Description of Algorithms

Previous studies have compared the performance of different pulse oximeters in the clinical environment[11] and in specially designed volunteer studies.[3,4,12] In recent years, new algorithms in pulse oximeters have improved their performance. The basic principles of the algorithm in the Ivy 2000 with Masimo SET technology have been described by Barker and Shah.[3]

The algorithm implemented in the Agilent device, according to Agilent, uses patented digital techniques in the frequency domain and an analysis of the resulting Fourier spectrum to remove noise from the signal. A fast Fourier transform generates Fourier spectral peaks, representing the pulse, the pulse harmonics, noise artifact, and any noise and artifact harmonics. Peaks from noise and artifact are eliminated by failure to pass a number of qualifying tests, leaving only the actual pulse and its harmonics. The tests base elimination on unrealistic S_{pO_2} values, pulse rate values, perfusion values, and statistical analysis of those values to eliminate nonsignal harmonics. Recently the latest Agilent technology (software version B.0) was tested against the Ivy 2000 with Masimo SET and the Nellcor N-3000 in a software simulation of critical clinical situations.[12]

The Datex-Ohmeda 3900P uses a feature called TruTrak data sampling, which detects and calculates S_{pO_2} 30 times per second. By calculating all along the waveform, it is not prone to motion-induced information gaps seen with oximeters that calculate S_{pO_2} after detecting the peak and trough of a clean pulse. TruTrak data sampling determines how well the red and IR waveforms match, and, if they are too dissimilar, the data period is identified and isolated from the calculations. S_{pO_2} data are compared with previous data points and weighted according to the degree of consistency. Inconsistent values are assigned lower weights and thus have less impact on the S_{pO_2} averaging calculation.

The Nellcor N-395 oximeter introduces multiple algorithms for calculating pulse rate and S_{pO_2}, along with arbitration procedures for choosing among the outputs of those algorithms. A basic pattern-matching algorithm is used to identify individual pulses to drive one of the pulse rate methods as well as the audible beep tone. A second algorithm, based on an adaptive comb filter, is used to track pulse rate in the presence of severe noise. This method functions by "locking on" to the rhythmic portion of the optical signal that is near the previously determined pulse rate. The first of 2 saturation algorithms uses a digital filter locked to the adaptive comb filter pulse rate to enhance the signal-to-noise ratio of the IR and red signals that are used to compute S_{pO_2}. A second S_{pO_2} algorithm uses least squares fitting to find the best estimate of S_{pO_2} derived from continuously sampled IR and red data. The Nellcor N-395 is a member of the fourth generation of Nellcor pulse oximeters, whereas the N-3000 belongs to the third generation.

Conclusions

The pulse oximeters tested in this study reliably measured S_{pO_2} when movement was introduced as an artifact and when perfusion was reduced in the sensor area. Only during the clinically extreme condition of combined low perfusion and motion artifact was the reliable detection of S_{pO_2} $<$ 62% for the various devices. During combined reduced perfusion and motion artifact, 3 of the tested pulse oximeters also proved reliable for showing pulse rate, whereas the Nellcor N-3000 and the Datex Ohmeda 3900P showed clear differences from the ECG-measured heart rate—a fact that must be considered in the clinic.

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Modification of a Critical Care Ventilator for Use During Magnetic Resonance Imaging

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INTRODUCTION: The unique electromagnetic environment of the magnetic resonance imaging (MRI) scanner presents particular problems for critically ill patients requiring mechanical ventilation during MRI. Most currently available MRI-compatible ventilators are limited in scope and function and thus may not be suitable for patients requiring high peak inspiratory pressure or flow. METHODS: To determine whether a standard critical care ventilator could be used under MRI conditions, we modified a Siemens Servo 900C by replacing the standard oxygen blender with an MRI-compatible blender. We then calibrated the ventilator and tested it on a mechanical lung during active MRI scanning at magnetic fields up to 1.5 tesla. After verifying adequate function, we used the ventilator to support 21 critically ill patients requiring mechanical ventilation during MRI. RESULTS: In all cases we found no alterations in ventilator performance resulting from the electromagnetic interference typical of an MRI scan. We also found no abnormalities in the alarm systems for fraction of inspired oxygen, high inspiratory pressure, or minute volume. Finally, we found no degradation of MRI image quality resulting from ventilator operation during test scanning. CONCLUSIONS: We conclude that with minor modifications the Siemens 900C ventilator can safely ventilate critically ill patients during MRI. *Key words: mechanical ventilation, magnetic resonance imaging, equipment, ventilators, MRI.* [Respir Care 2002;47(1):61–68]

Introduction

Since the first description of normal human magnetic resonance imaging (MRI) in 1981,[1] the use of MRI has increased dramatically. MRI images have been particularly valuable for detecting abnormalities in the brain, spinal cord, spine, and major joints not readily apparent by conventional imaging techniques.

For mechanically ventilated patients, the electromagnetic environment of the MRI scanner raises several technical issues regarding safety and monitoring. The strong magnetic and radio frequency fields produced by the MRI scanner, for example, have been hypothesized to prevent the electronic components of most critical care ventilators from functioning properly.[2] Electromagnetic interference from intensive care ventilators may also interfere with the MRI process, potentially producing artifacts in the finished scans.[3,4] Finally, the strong magnetic field generated by the MRI magnet raises the possibility that the ventilator could be physically drawn toward the magnet and thus present a projectile hazard to the patient or to care personnel.[2]

Abnormalities in ventilator monitoring and alarm systems induced by electromagnetic interference may also increase the risk of patient harm. In pressure-control mode, tidal volume (V_T) varies because of changes in lung compliance or airways resistance, so V_T must be precisely monitored to avoid catastrophic hypoventilation. A malfunction of critical alarm or monitoring functions because of electromagnetic interference from the MRI scanner could be disastrous.

Some ventilators have been designed specifically for use during MRI,[5] but most of them lack the flexibility in mode and air flow characteristics, sophisticated monitoring and alarm systems, flow delivery with high airway pressures, and familiarity of operation typical of standard critical care ventilators. For patients who require high flows or pressures to maintain adequate gas exchange, use of a critical care ventilator during MRI scanning would provide a greater margin of safety throughout the procedure

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and possibly reduce errors resulting from unfamiliarity with the ventilators designed for MRI use. We therefore systematically evaluated the Siemens Servo 900C ventilator for use under electromagnetic conditions typical of the MRI environment. We then evaluated ventilator performance in the MRI suite, using a test lung and with critically ill patients during active scanning conditions.

Methods

We used a Servo 900C ventilator (Siemens Medical Systems, Danvers, Massachusetts) equipped with standard ventilator circuits (Allegiance Healthcare, McGaw Park, Illinois) in all experiments. Bench testing was performed in the Respiratory Therapy Laboratory at the University of Chicago, using a test lung (model 1600i, Michigan Instruments, Grand Rapids, Michigan) and a Wright spirometer (Ferraris Medical, Holland, New York). Magnetic fields for bench testing were produced by a common handheld magnet. In the MRI scanner, the ventilator was tested using the ventilator's on-board monitoring system and double-checked for gross error with a spirometer (Puritan Bennett MA-1, Tyco Healthcare Group, Pleasanton, California). Fraction of inspired oxygen (F_{IO_2}) was checked with an oxygen analyzer (Mini-Ox, Mine Safety Appliance Company, Pittsburgh, Pennsylvania) during calibration outside the MRI suite and the Siemens on-board oxygen analyzer inside the suite.

Bench Testing

To prepare the ventilator for operation under MRI conditions, we first identified all ferromagnetic ventilator components by running a small magnet across all portions of the ventilator. We identified circuit board elements, wiring busses, bellows springs, and the standard oxygen blender as the only magnet-reactive components. We then replaced the oxygen blender with a Bird MRI microblender (part #10030A, Bird Products [VIASYS], Palm Springs, California) equipped with a brass (nonferrous) fitting. The circuit boards, wiring elements, and bellows springs were not replaced. Disposable plastic ventilator circuits (Allegiance Healthcare, McGaw Park, Illinois) were then modified to produce an adult and a pediatric/neonatal configuration capable of reaching the patient inside the magnet. Standard 6-foot adult circuits (22 mm inner diameter [ID]) and 4- foot pediatric (15 mm ID) or neonatal (10 mm ID) circuits were lengthened by using a 22 mm adapter and connecting appropriate extensions to produce a uniform 9-foot circuit length. Heating wires built into the pediatric circuits were removed and the wire port was capped.

Bench Calibration

After modification, the ventilator was calibrated in our lab using the standard Siemens calibration protocol. First, inspiratory and expiratory flow transducers and all mechanical manometers were zeroed with no gas flow in the system. To calibrate inspiratory and expiratory pressure transducers, the working pressure was set to 60 cm H_2O, the inspiratory and expiratory limbs were connected, and the on-board pressure manometer reading was compared to the digital monitor of airway pressure. The inspiratory pressure transducer was calibrated using the Siemens calibration manometer (Siemens Medical Systems, Danvers, Massachusetts) at values of 45–50 cm H_2O. To calibrate the expiratory minute volume ($\dot{V}$) meter, preset inspiratory $\dot{V}$ was set to 7.5 L/min and the on-board expiratory $\dot{V}$ meter and digital monitor were checked to verify the same values. Set V_T was compared to values obtained from the on-board measurement system at different preset V_T. A Wright spirometer was used to confirm that there was no gross error in volume and flow measurements. Positive end-expiratory pressure (PEEP) was then set at 10 cm H_2O and checked using the on-board pressure manometer. The ventilator was observed for self-triggering behavior while in volume-control mode and continuous positive airway pressure mode. Finally, the oxygen blender was set to 20.9% and then to 100% and compared to the readings from the ventilator's on-board oxygen analyzer, and the Mini-Ox analyzer was placed in the inspiratory limb.

MRI Suite Testing

We then modified the MRI scanner to accommodate the requirements of the Siemens ventilator. Testing was performed in the MRI suite at the University of Chicago, using a General Electric Sigma Horizon LX, 5x platform, MRI system (General Electric Medical Systems, Milwaukee, Wisconsin). To provide a source of compressed air inside the MRI chamber, a portable air compressor was stationed outside the MRI suite and a 20–25-foot custom high-pressure compressed air hose was fed under the floor and through an access panel for oxygen located next to the magnet. The ventilator was then moved into the MRI suite and plugged into an electrical outlet. The ventilator was positioned 2–3 feet away from the scanner and the wheels on the ventilator stand were locked. The oxygen inlet on the ventilator was connected to an oxygen wall outlet (50 psi), and the air inlet was connected to the custom compressed air hose. The plastic test lung was attached to the Y-piece of the ventilator circuit and the ventilator was powered on. The MRI scanner's automatic pre-scanning routine was then activated, with General Electric engineers observing the automatic calibration and shimming routines, while the ventilator was operating. Calibration and

shimming procedures were then rechecked manually for comparison. A test scan was then performed on both the plastic test lung and a test object provided by General Electric, which were situated in the scanner where the patient head and torso would be positioned. Test scans were repeated with the ventilator off, on, and at 1, 2, 3, and 4 feet from the magnet, to evaluate the effect of distance on ventilator function and MRI artifact.

We then examined the operation of the inspiratory scissors valve, step motor, and flow transducer, which were connected to pre-amp assembly cables found to have a magnetic response. Under conditions of various set $\dot{V}$, we monitored set $\dot{V}$, actual expired $\dot{V}$, and both inspired and expired V_T. To confirm that V_T measured by the ventilator's on-board monitoring system was correct, we connected the MA-1 volume spirometer to the exhalation valve while the test scan was in progress. Although the MA-1 is a relatively inaccurate device, we used it for MRI compatibility reasons and to confirm on-board measurements. To verify that inspiratory flows were regulated appropriately, we reduced the set inspiratory time percentage from 33% to 25%, while holding $\dot{V}$ and respiratory rate constant, and measured the effect on V_T.

We then examined the operation of the expiratory scissors valve, pull magnet, and flow transducer during active scanning. Because the pre-amp assembly for the expiratory flow transducer was ferromagnetic during bench testing, we compared set PEEP levels to measurements obtained with the on-board manometer. PEEP levels were tested at 2.5, 5, 7.5, 10, 12, 15, and 20 cm H_2O.

Monitoring systems for F_{IO_2}, expired $\dot{V}_E$, and peak inspiratory pressure were then tested during scanning. Oxygen monitoring was tested by setting the Bird blender at 21, 30, 40, 60, 80, and 100% oxygen and waiting 5 minutes at a $\dot{V}_E$ of 7.5 L/min and a respiratory rate of 20 breaths/min. The F_{IO_2} measured by the on-board oxygen analyzer was then compared to the set F_{IO_2} during active scanning. Oxygen alarms were tested at 30, 40, 60, and 80% oxygen, with the high and low alarms 10% above and below at each value. The low F_{IO_2} alarm was tested by disconnecting the oxygen supply line to the ventilator, and the high F_{IO_2} alarm was tested by disconnecting the air supply line.

The expired $\dot{V}$ alarm was tested during active scanning by setting the $\dot{V}$ control to 7.5 L. The low expired $\dot{V}$ alarm was then evaluated by setting the low expired $\dot{V}$ alarm to 7 L/min and disconnecting the temperature port adapter to simulate an air leak. The high expired $\dot{V}$ alarm was tested by setting the high expired $\dot{V}$ alarm for 7.7 L/min and repeatedly manipulating the plastic test lung to cycle the ventilator in volume-control mode.

The high inspiratory pressure alarm was tested by preventing excursion of the plastic test lung during inspiration, to force airway pressures above set limits. Inspiratory pressure alarms were tested at 30, 40, 50, 60, 70, and 80 cm H_2O, with the alarm limit set 10 cm H_2O above the set pressure.

All measures of ventilator alarm function using the mechanical test lung were performed 3 times to verify repeatability, and a minimum of 3 minutes were allowed to elapse between changes in ventilator settings.

Use in Critically Ill Patients

The Siemens 900C ventilator has been used for over 24 months at our institution to support critically ill patients during MRI scanning. With approval from the institutional review board and in accordance with hospital guidelines, we reviewed charts of patients ventilated in this manner, to confirm the effectiveness of the Servo 900C ventilator in this environment. Chart review included age, gender, oxygen saturation during MRI scanning, and level of sedation. In addition, we reviewed the attending radiologist's interpretation of each MRI with respect to the quality of image or the presence of unusual artifacts possibly resulting from the presence of the ventilator.

In all cases, the respiratory therapy department was notified of the need for mechanical ventilation at the time the MRI scan was scheduled. Prior to transporting the patient, the Siemens ventilator would be moved into the MRI suite, attached to electricity and compressed gas sources, and checked with a plastic test lung for normal function. The patient would then be transferred from intensive care to the MRI unit while using a resuscitation bag, portable oxygen tank, and monitoring devices. Since the portable oxygen tank was ferromagnetic, extension tubing was added to allow the resuscitation bag to reach the patient inside the MRI scanner. Stable vital signs were verified prior to and during the MRI scan.

Results

Bench Testing

Figure 1 and Table 1 show the ventilator parts that, on initial evaluation with a handheld magnet, were found to have a magnetic response.

Bench Calibration

We modified the ventilator for MRI use by replacing the oxygen blender with a Bird MRI microblender. Bench calibration found the following volume factors, volume calibration measurements, and F_{IO_2} measurements.

Volume Factors for Elongated Ventilator Circuits. Lost volume factors were calculated for each of the modified circuits: adults 3.0 mL/cm H_2O; pediatric 1.1 mL/cm H_2O; neonates 0.7 mL/cm H_2O.

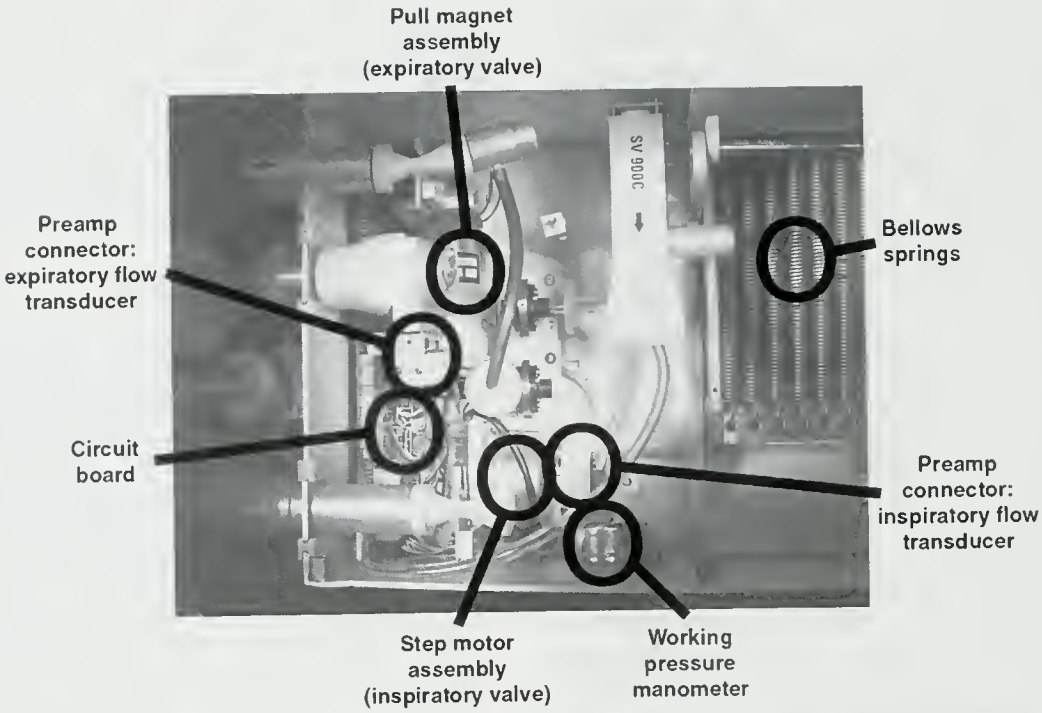

Fig. 1. Interior of the Siemens Servo 900C ventilator, indicating parts found to have a magnetic response.

Table 1. Magnetic Response of Components of the Siemens Servo 900C Ventilator*

Ventilator Subsection	+ Magnetic Response	− Magnetic Response
Outer shell, stand, circuit support arm	Wheel locks	Ventilator enclosure
	Circuit support arm	Ventilator stand
Pneumatic section	Inspiratory and expiratory flow transducer assembly	Inspiratory scissors valve
	Inspiratory and expiratory cable connector	Expiratory scissors valve
	Bellows springs	All other components, including hoses, bellows, and valves
	Step motor assembly	
	Pull magnet assembly	—
Electronic control section	Circuit boards	—
	Wire connectors	—
	Input/output connectors	—
Blender	Enclosure and contents	—
Ventilator circuit assembly	—	All other components

*On bench testing. Only the blender components required replacement with nonmagnetic components. We removed the Servo 900C oxygen blender and replaced it with a Bird MRI microblender

Volume Calibration. The modified ventilator performed as expected during normal volume calibration. Correlation between set V_T and measured inspiratory V_T was 0.997 for the on-board volume monitor and 1.0 for the test lung. Correlation between set V_T and measured expiratory V_T was 0.998 for the on-board volume monitor and 0.997 for the Wright spirometer. (Table 2).

Fraction of Inspired Oxygen. Correlation between set F_{IO_2} levels and values measured was 0.999 for the Siemens on-board system and 0.998 for the external Mini-Ox oxygen analyzer (Table 3).

MRI Suite Testing

Effect of Ventilator Placement on MRI Scanning Variables. The presence of the ventilator in the MRI suite produced a minor alteration in the center frequency, or "shim" of the magnet. This alteration was readily compensated for automatically by the MRI system's normal

Table 2. Volume Calibration Measurements from Bench Testing of the Modified Servo 900C*

Transducers	Set V (L/min)	f (breaths/min)	Servo 900C (mL)	Test Lung (mL)	Spirometer (mL)
Inspiratory	10.0	20	490	500	—
	12.0	20	600	600	—
	14.0	20	685	700	—
Expiratory	10.0	20	479	—	470
	11.9	20	590	—	595
	13.9	20	680	—	695

*Modification consisted of replacing the Servo 900C oxygen blender with the Bird MRI microblender. Calibration was performed at 3 V/f settings, with inspiratory and expiratory tidal volumes measured with the Servo 900C and confirmed with the test lung and the spirometer.
V = minute volume
f = respiratory frequency

pre-scanning routine and was confirmed as minor by the General Electric engineers, who reproduced the automatic adjustment manually. MRI scans of a test object provided by General Electric were identical with the ventilator positioned 1, 2, 3, and 4 feet from the magnet, further suggesting that the ventilator's effect on the MR image was minor. No artifacts from ventilator alarms were observed.

Flow Transducer Measurements During MRI Scanning. At a rate of 20 breaths/min and set $\dot{V}$ of 6–20 L, correlation between set V_T and values measured with either the on-board volume monitor or the MA-1 spirometer exceeded 0.999. We observed no malfunction in either inspiratory or expiratory flow transducer measurements during active scanning. Correlation between V_T measurements by the MA-1 spirometer and the on-board expiratory flow transducer also exceeded 0.999 (Table 4).

Effect of the MRI Suite on the Oxygen Sensor. Correlation between the blender F_{IO_2} setting and the Servo 900C on-board oxygen analyzer during active MRI scanning exceeded 0.998 (see Table 3).

Alarm Testing. We found no abnormalities during repeated testing of high and low F_{IO_2}, expired $\dot{V}$, or high inspiratory pressure alarms during MRI scanning.

Auto-Triggering. We observed no self-triggering behavior during either volume-control mode or continuous positive airway pressure mode during the test scan.

Use in Human Patients. Table 5 shows the clinical and demographic information on 21 patients successfully ventilated with the Servo 900C during MRI. All patients were accompanied by a nurse or physician and transferred from manual ventilation to the Siemens ventilator by a respiratory therapist. Intensive care unit ventilator settings were duplicated during MRI scanning, and vital signs were recorded by the nurse or physician.

Two untoward events occurred with use of the Servo 900C ventilator during MRI scanning. Both occurred during setup and prior to actual use with patients, and neither was referable to the MRI environment. In one case, the ventilator was found to deliver 25 cm H_2O PEEP while being set for 5 cm H_2O PEEP. This malfunction was traced to a twisted rubber expiratory valve, which was replaced. In another case, the ventilator was found to deliver 35 cm H_2O PEEP while being set for 5 cm H_2O PEEP. In this case the expiratory flow transducer was found to be malfunctioning during calibration, prior to placing the ventilator into the MRI scanner. No projectile risks were encountered. Inadvertent disconnection from the ventilator occurred in 8 patients; these events were readily detected and underscore the importance of operative ventilator alarm systems during scanner table transport.

Discussion

Magnetic resonance imaging is based on signals emitted from hydrogen nuclei (protons) within the body. During the MRI exam, the patient is positioned in a tube surrounded by a large superconducting magnet that produces

Table 3. Comparison of Set F_{IO_2} and Oxygen Values Measured by the Servo 900C On-Board Analyzer and the Mini-Ox Device*

				F_{IO_2} (%) During Bench Testing					
Blender setting	21	30	40	50	60	70	80	90	100
Servo 900C on-board oxygen analyzer	20.9	28.9	39	50	58.9	71	82	89.9	97.8
Mini-Ox	20.9	29	40	50	59.5	70.3	80	91	98.3
				F_{IO_2} (%) During MRI Scanning					
Blender setting	21	30	40	55	60	75	80	90	100
Servo 900C on-board oxygen analyzer	20.9	29.8	41.5	56	59.1	74.5	80	90	98.5

F_{IO_2} = fraction of inspired oxygen
*Values were obtained during bench testing and in the MRI scanner during MRI imaging with the modified ventilator

Table 4. Volume Measurements During Active Scanning*

Flow Transducer	Pre/Post $\dot{V}$ (L/min)	f (breaths/min)	V_T (mL) Servo 900C	V_T (mL) MA-1 Spirometer
Inspiratory	4.0	20	200	—
Expiratory	4.0	20	198	200
Inspiratory	6.0	20	300	—
Expiratory	6.0	20	308	300
Inspiratory	7.5	20	375	—
Expiratory	7.5	20	380	380
Inspiratory	10.0	20	500	—
Expiratory	10.0	20	495	500
Inspiratory	16.0	20	795	—
Expiratory	16.0	20	805	800
Inspiratory	20.0	20	990	—
Expiratory	20.0	20	998	1000

*Tidal volume (V_T) was measured using the on-board volume monitor and confirmed using the MA-1 spirometer
$\dot{V}$ = minute volume
f = respiratory frequency

Table 5. Clinical and Demographic Information*

Patient #	Age	Sex	S_{pO_2} (%) during MRI	Mental Status	Artifacts in Scan
1	44	F	96	calm	none
2	56	F	95	calm	none
3	3	F	99	sedated	none
4	6	M	98	sedated	none
5	39	F	99	calm/sedated	none
6	2	M	98	calm/sedated	none
7	8	M	96	lightly sedated	none
8	81	M	95	lightly sedated	none
9	39	F	99	heavily sedated	none
10	18	F	99	calm/sedated	none
11	35	M	96	sedated	none
12	17	M	95	lightly sedated	none
13	26	F	99	calm	none
14	40	M	99	lightly sedated	none
15	2	M	98	lightly sedated	none
16	40	M	97	moderately sedated	none
17	74	M	98	calm	none
18	61	M	92	sedated	none
19	8	M	99	lightly sedated	none
20	6	M	99	sedated	none
21	66	M	96	sedated	none

*From 21 patients ventilated with the Servo 900C during magnetic resonance imaging (MRI).
S_{pO_2} = oxygen saturation measured via pulse oximetry

an intense magnetic field around the patient. This magnetic field causes the spin of all the hydrogen nuclei (protons) in the body to align. To produce the image, the MRI scanner emits a strong radio frequency pulse that briefly knocks the protons out of alignment. As the protons realign, they emit a weak radio signal detectable by the MRI scanner. The strength of signal depends on the concentration of the protons in different body tissues, which correlates strongly with tissue water content. The MRI scanner then combines signals from the different tissues in the region of interest into an image of the desired structure.

The combination of the intense magnetic field and radio frequency pulses generated by an MRI scanner creates a unique electromagnetic environment that can interact substantially with complex life-support machinery. Electrically powered devices such as pacemakers, infusion pumps, and ventilators may malfunction as a result of electromagnetic interference, but also interfere with the completed MRI image. Patients have even undergone surgery based on MRI artifacts produced by medical devices,[5] underscoring the importance of MRI-device interactions.

Because of the superior ability of MR images to detect abnormalities in brainstem and other central nervous system structures, MR scanning is particularly useful in critically ill patients undergoing neurologic evaluation. Many of these patients have severe lung disease and require mechanical ventilation with high peak inspiratory pressure or nonstandard modes. For those patients the MRI environment presents 3 important technical concerns. First, electromagnetic interference may cause the ventilator to malfunction. Regulation of inspiratory gas mixture and flow, inspiratory and expiratory timing, and patient triggering is typically electronic in modern ventilators and may be disrupted by strong magnetic or radio frequency fields. Second, ventilator alarm and monitoring systems may be compromised by electromagnetic interference. Failure of alarm systems may result in catastrophe, particularly when complex ventilation modes are used and the patient is located deep within the magnet. Finally, electromagnetic interference produced by the ventilator may produce MRI artifacts.

Although in the past clinicians have hand-ventilated patients throughout the procedure,[6] such a strategy is difficult with prolonged scanning or in a patient with severe lung disease. The use of 20-foot breathing circuit extensions with the ventilator stationed outside the MRI suite has been described.[7] MRI-compatible ventilators have been developed but are usually transport ventilators, limited in scope and function. Examples include the Omni-Vent (Allied Health Care, St Louis, Missouri), Bio-Med vent (Bio-Med Devices, Guilford, Connecticut), and the RespirTech PRO (Vortran Medical Technologies, Sacramento, California). With the exception of the Bio-MED ventilator, these ventilators operate in pressure-control mode only, where exhaled V_T depends on patient compliance and set inspiratory time. In addition, monitoring and alarm options may be limited.

Another drawback to using those types of ventilators during MRI scanning is that, with the exception of the RespirTech PRO, they use a single-limb breathing system: the same gas path is used for both inspiration and expiration, increasing the likelihood of mixing inspiratory and expiratory gases with improper location of the exhalation valve in the circuit.

Finally, there is substantial risk in switching critically ill patients between different ventilators that may not support the same modes. Many critically ill patients require specialized ventilator settings and can deteriorate when ventilated with different equipment or in different modes. For patients in whom transport to and from the MRI scanner represents increased risk, using the same ventilator in the intensive care unit and the MRI suite avoids possible errors in transferring complex ventilator settings.

We found that replacing the Servo 900C oxygen blender with the Bird MRI microblender allowed the Servo 900C to function normally in the MRI electromagnetic environment, and that the ventilator did not produce MR image artifacts. Although other components of the ventilator had a magnetic response on bench testing (see Table 1), replacing the blender was the only modification required to allow normal function in the MRI environment. When tested during active scanning, we found no abnormalities in the ventilator's ability to function in volume-control mode and continuous positive airway pressure mode, and we verified that monitoring of $\dot{V}_E$, F_{IO_2}, inspiratory pressure, PEEP, and gas flows was unaltered. No artifact or "ghost" images were noted on MRI scans completed by General Electric engineers. Finally, we observed that the ventilator was able to alarm properly when $\dot{V}$, F_{IO_2}, and high inspiratory pressure alarm levels were exceeded during active scanning.

The presence of the ventilator in the MRI suite required a background adjustment in the center frequency or "shim" of the MRI magnet, but this adjustment was minor, was made automatically during the MRI system's pre-scanning, and was confirmed as appropriate by the General Electric engineers. Shim adjustments were readily performed automatically with the ventilator placed 1, 2, 3, and 4 feet from the magnet, suggesting that electromagnetic interference generated by the ventilator was not excessive. Scans of a test object provided by General Electric were identical with varying ventilator placement, further evidencing the relatively minor effect of ventilator-produced interference.

Although we believe that our modified Servo 900C ventilator is safe in an MRI environment, we also recommend that on-site testing be performed with every ventilator being considered for use in an MRI suite. Differences in manufacturing tolerances between individual ventilators, as well as between different MRI suites, may limit the generalization of our data. Until a critical care ventilator is produced that is explicitly labeled MRI-compatible, we believe that thorough and systematic testing of ventilator function and alarm and monitoring systems followed by "on-line" testing with a test lung during actual scanning is the best way to guarantee safe ventilation in critically ill patients during MRI.

Conclusions

We conclude that a modified Siemens Servo 900C ventilator is capable of safely and effectively delivering mechanical ventilation to critically ill patients during MRI. We recommend the following minor changes to the Servo 900C critical care ventilator:

1. Replace the on-board gas blender with an MRI-safe blender such as the Bird MRI microblender (part #10030A, Bird Products) equipped with a brass fitting.
2. Remove the circuit support arm prior to moving the ventilator into the suite.
3. Lock the wheels on the cart during the procedure.
4. Avoid coiling the electrical cord, which can facilitate electrical currents induced by the magnetic field.
5. Because of the increased compliance associated with the increased ventilator circuit length, we recommend the use of pressure-control ventilation in small infants or when V_T is < 100 mL.
6. A heat and moisture exchanger should be used for long cases and the ventilator stationed at least 2–3 feet from the MRI scanner to avoid projectile risk.
7. Expired $\dot{V}$ and peak inspiratory pressure should be carefully monitored, as the patient will often be deep inside the scanner and abnormalities in ventilation will be difficult to diagnose.
8. Caregivers should position themselves in order to be able to hear ventilator alarms.
9. Aluminum oxygen cylinders should be used in MRI suites.

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Evidence-Based Guidelines for Weaning and Discontinuing Ventilatory Support

A Collective Task Force Facilitated by the American College of Chest Physicians, the American Association for Respiratory Care, and the American College of Critical Care Medicine

Introduction
Pathophysiology of Ventilator Dependence
Criteria to Assess Ventilator Dependence
Managing the Patient Who Has Failed a Spontaneous Breathing Test
Role of Tracheotomy in Ventilator-Dependent Patients
The Role of Long-Term Facilities

[Respir Care 2002;47(1):69–90]

Introduction

The discontinuation or withdrawal process from mechanical ventilation is an important clinical issue.[1,2] Patients are generally intubated and placed on mechanical ventilators when their own ventilatory and/or gas exchange capabilities are outstripped by the demands placed on them from a variety of diseases. Mechanical ventilation also is required when the respiratory drive is incapable of initiating ventilatory activity, either because of disease processes or drugs. As the conditions that warranted placing the patient on the ventilator stabilize and begin to resolve, attention should be placed on removing the ventilator as quickly as possible. Although this process often is termed "ventilator weaning" (implying a gradual process), we prefer the more encompassing term "discontinuation."

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Unnecessary delays in this discontinuation process increase the complication rate from mechanical ventilation (eg, pneumonia, airway trauma) as well as the cost. Aggressiveness in removing the ventilator, however, must be balanced against the possibility that premature discontinuation may occur. Premature discontinuation carries its own set of problems, including difficulty in reestablishing artificial airways and compromised gas exchange. It has been estimated that as much as 42% of the time that a medical patient spends on a mechanical ventilator is during the discontinuation process.[3] This percent is likely to be much higher in patients with more slowly resolving lung disease processes.

There are a number of important issues involved in the management of a mechanically ventilated patient whose disease process has begun to stabilize and/or reverse such that the discontinuation of mechanical ventilation becomes a consideration. First, an understanding of all the reasons that a given patient required a mechanical ventilator is needed. Only with this understanding can medical management be optimized. Second, assessment techniques to identify patients who are capable of ventilator discontinuation need to be utilized. Ideal assessment techniques should be able to easily and safely distinguish which pa-

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Table 1. Grades of Evidence

Grade	Description
A	Scientific evidence provided by well-designed, well-conducted, controlled trials (randomized and nonrandomized) with statistically significant results that consistently support the guideline recommendation
B	Scientific evidence provided by observational studies or by controlled trials with less consistent results to support the guideline recommendation
C	Expert opinion supported the guideline recommendation, but scientific evidence either provided inconsistent results or was lacking

tients need prompt discontinuation and which need continued ventilatory support. Third, ventilator management strategies for stable/recovering patients who still require some level of ventilatory support need to be employed. These strategies need to minimize both complications and resource consumption. Fourth, extended management plans (including tracheotomy and long-term ventilator facilities) need to be considered for the long-term ventilator-dependent patient.

To address many of these issues, the Agency for Healthcare Policy and Research (AHCPR) charged the McMaster University Evidence Based Practice Center to do a comprehensive evidence-based review of many of the issues involved in ventilator weaning/discontinuation. Led by Deborah Cook MD, an exhaustive review of several thousand articles in the world literature resulted in a comprehensive assessment of the state of the literature in 1999.[4] At the same time, the American College of Chest Physicians (ACCP), the Society for Critical Care Medicine (SCCM), and the American Association for Respiratory Care (AARC) formed a task force to produce evidence-based clinical practice guidelines for managing the ventilator-dependent patient during the discontinuation process. The charge of this task force was to utilize the McMaster AHCPR report as well as their own literature review to address the following 5 issues: (1) the pathophysiology of ventilator dependence; (2) the criteria for identifying patients who are capable of ventilator discontinuation; (3) ventilator management strategies to maximize discontinuation potential; (4) the role of tracheotomy; and (5) the role of long-term facilities. Review/writing teams were formed for each of these issues.

From these evidence-based reviews, a series of recommendations were developed by the task force, which are the basis of this report. Each recommendation is followed by a review of the supporting evidence, including an assessment of the strength of the evidence (Table 1). As there were many areas in which evidence was weak or absent, the expert opinion of the task force was relied on to "fill in the gaps." Consensus was reached, first, by team discussions and, later, through the repeated cycling of the draft through all members of the task force.

Both the McMaster AHCPR group and the task force recognized the needs for the future. These include more randomized controlled trials to look at a number of issues. Among the more important questions that need answering are the following: (1) Which criteria are the best indicators of reversal of respiratory failure in the screening process? (2) What factors are involved in ventilator dependence and which measurement techniques are most useful in determining ultimate success in the discontinuation process? (3) In balancing discontinuation aggressiveness against the risks of premature discontinuation, what is a reasonable reintubation rate in patients recently removed from ventilatory support? (4) What is the value of trying to reduce levels of partial ventilator support in stable/recovering patients who have failed a discontinuation assessment? (5) What role do tracheotomies have in facilitating the discontinuation process? (6) What is the role of the long-term facility, and when should patients be transferred to such facilities?

Pathophysiology of Ventilator Dependence

Introduction

Patients require mechanical ventilatory support when the ventilatory and/or gas exchange capabilities of their respiratory system fail. This failure can be the result of processes both within the lung as well as in other organ systems, most notably the central nervous and the cardiovascular systems. Although patients may be dependent on ventilatory support for brief periods of anesthesia or neuromuscular blockade, the term "ventilator-dependent" is usually reserved for patients with a need for mechanical ventilation beyond 24 hours or by the fact that they have failed to respond during discontinuation attempts. Under these circumstances, the clinical focus should be not only on ventilator management but also should include a search for all of the possible reasons (especially potentially reversible ones) that may explain the ventilator dependency.

Recommendation 1. *In patients requiring mechanical ventilation for > 24 hours, a search for all the causes that may be contributing to ventilator dependence should be undertaken. This is particularly true in the patient who has failed attempts at withdrawing the mechanical ventilator. Reversing all possible ventilatory and nonventilatory issues should be an integral part of the ventilator discontinuation process.*

Table 2. Causes of Ventilator Dependency

Causes	Description
Neurologic controller	Central drive; peripheral nerves
Respiratory system	Mechanical loads: respiratory system mechanics; imposed loading
	Ventilatory muscle properties: inherent strength/endurance; metabolic state/nutrients/oxygen delivery and extraction
	Gas exchange properties: vascular properties and ventilation/perfusion matching
Cardiovascular system	Cardiac tolerance of ventilatory muscle work; peripheral oxygen demands
Psychological issues	

Evidence (Grade B)

There are a number of specific reasons why patients may be ventilator-dependent (Table 2). Determining which factor or factors may be involved in a given patient requires both clinical awareness of these factors as well as focused clinical assessments. The search for the underlying causes for ventilator dependence may be especially important if previously unrecognized, but reversible, conditions are discovered.

Neurologic Issues: The ventilatory pump controller in the brainstem is a rhythm and pattern generator, which receives feedback from cortical, chemoreceptive and mechanoreceptive sensors. The failure of this controller can come from several factors.[5–12] These factors can be either structural (eg, brainstem strokes or central apneas) or metabolic (eg, electrolyte disturbances or sedation/narcotic usage[13,14]). The failure of the peripheral nerves also can be the result of either structural factors[15] or metabolic/drug factors.[16,17] A unique neurologic dysfunction that also could cause ventilator dependence is obstructive sleep apnea, in which an artificial airway may be necessary to maintain airway patency.[10,11]

Respiratory System Muscle/Load Interactions: Often, patients who exhibit ventilator dependence do so because there appears to be a mismatch between the performance capacity of ventilatory pump and the load placed on it (ie, the capacity/load imbalance hypothesis).[18–23] There is ample evidence that ventilatory pump performance may be impaired in ventilator-dependent patients because ventilatory muscles are weak. This may be a consequence of atrophy and remodeling from inactivity.[2,24] It may also be a consequence of injury from overuse and of insults associated with critical illness neuropathy and myopathy.[25–29] A number of drugs (eg, neuromuscular blockers, aminoglycosides, and corticosteroids) also can contribute to myopathy,[17,30–32] as can various metabolic derangements (see below). Finally, dynamic hyperinflation can put ventilatory muscles in a mechanically disadvantageous position.[33] In a number of studies, patients who failed to respond to a withdrawal from mechanical ventilation tended to be weaker (ie, they had a lower performance capacity) than those who succeeded,[34–49] but, in general, the within-group variability in respiratory muscle strength was too large to justify general conclusions.

Ventilatory muscle fatigue also could contribute to poor muscle performance. However, the role of fatigue in ventilator dependence is not well understood, and the studies performed to date[21,26,50–54] have failed to delineate the sensitivity and specificity of specific fatigue tests in ventilator-dependent patients. Ventilatory support reduction-related changes in transdiaphragmatic pressure, respiratory rate, and thoracoabdominal dyssynchrony are clearly not specific manifestations of respiratory muscle fatigue.[55–60] The most promising diagnostic test of diaphragm contractility to date is the transdiaphragmatic pressure measurement during twitch stimulation of the phrenic nerves.[21,61] However, too few patients have been studied with this technique to draw any meaningful conclusions about the prevalence of diaphragm fatigue that is attributable to ventilator dependence.

The load on the ventilatory muscles is a function of ventilation demands and respiratory system mechanics (ie, primarily compliance and resistance). Normal minute ventilation during spontaneous breathing is generally < 10 L/min, normal respiratory system compliance (ie, tidal volume/static inflation pressure) is generally $> 50–100$ mL/cm H_2O, and normal airway resistance (ie, peak-static inflation pressure/constant inspiratory flow) is generally $< 5–15$ cm H_2O/L/s. Ventilation demands can increase as a consequence of increased oxygen demands in patients with sepsis or increased dead space in patients with obstructive diseases. Compliance worsening can be a consequence of lung edema, infection, inflammation, or fibrosis, and of chest wall abnormalities such as edema or surgical dressings. Resistance worsening can be a consequence of bronchoconstriction and airway inflammation. Additional load can also be imposed by narrow endotracheal tubes and by insensitive or poorly responsive ventilator demand valves.

The load imposed by ventilation demands interacting with respiratory system mechanics can be expressed as respiratory work, pressure time integral, or the change in metabolism (eg, the oxygen cost attributable to breathing). Many studies[19,35,62–66] show that patients who are ventilator-dependent tend to have larger respiratory muscle loads than do patients who can be withdrawn from mechanical ventilation. In patients with airways obstruction, the load imposed by dynamic hyperinflation has received particular attention as an important contributor to ventilator depen-

dence.[23,33,65,67–71] As is true for measures of ventilatory pump capacity, however, most investigators report considerable overlap in load parameters between patients with different discontinuation outcomes.

Patients who go on to fail to respond to ventilator withdrawal attempts because of a capacity/load imbalance tend to display rapid, shallow breathing patterns.[2,72,73] This pattern is advantageous from an energetics perspective, but it is also associated with increased dead space and wasted ventilation and, hence with impaired carbon dioxide elimination. Chemoreceptive and mechanoreceptive feedback into the neural control of breathing is not well understood, and thus it is difficult to distinguish whether this breathing pattern is a consequence of a reduced respiratory drive per breath or an inability of ventilatory muscles to respond to an appropriately increased neural stimulus.[19,62,65,71,72]

Metabolic Factors and Ventilatory Muscle Function: Nutrition, electrolytes, hormones, and oxygen transport are all metabolic factors that can affect ventilatory muscle function. Inadequate nutrition leads to protein catabolism and loss of muscle performance.[74,75] The normal hypoxic ventilatory response and the hypercapnic ventilatory response also have been shown to deteriorate under conditions of semistarvation.[76] In contrast, overfeeding also can impair the ventilator withdrawal process by leading to excess carbon dioxide production, which can further increase the ventilation loads on ventilatory muscles. Studies[77,78] have suggested that proper nutritional support can increase the likelihood of success of ventilator withdrawal. A number of electrolyte imbalances also can impair ventilatory muscle function.[5,9,79–81] Phosphate deficiency has been associated with respiratory muscle weakness and ventilator withdrawal failure. A study demonstrating improved transdiaphragmatic pressure values with the repletion of serum phosphorus levels in patients receiving mechanical ventilation, however, did not specifically address the issue of ventilator withdrawal.[79] Magnesium deficiency also has been reported to be associated with muscle weakness,[82] although the relationship to ventilator dependence has not been specifically addressed. Finally, bicarbonate excretion from inappropriate overventilation (often occurring in patients with chronic obstructive pulmonary disease (COPD) with chronic baseline hypercapnia) can impair ventilator withdrawal efforts as the patient has a diminished capacity to compensate for hypercapnia. Severe hypothyroidism and myxedema directly impair diaphragmatic function and blunt ventilatory responses to hypercapnia and hypoxia.[83,84] Other hormonal factors that are important for optimal muscle function include insulin/glucagon and adrenal corticosteroids.

As in other organs, adequate oxygen delivery and oxygen uptake by the ventilatory muscles is necessary for proper muscle function.[85,86] Impaired oxygen delivery can be a consequence either of inadequate oxygen content or of inadequate cardiac output.[87] Impaired oxygen uptake occurs most commonly during systemic inflammatory syndromes such as sepsis.[88]

Gas Exchange Factors: Gas exchange abnormalities can develop during ventilatory support reductions for several reasons. Various lung diseases produce ventilation-perfusion imbalances and shunts. Ventilator dependence thus may be a consequence of a need for high levels of expiratory pressure and/or the fraction of inspired oxygen (F_{IO_2}) to maintain an adequate oxygen content.[5,9] A patient with hypoxemia also can develop a fall in mixed venous P_{O_2} levels from the cardiovascular factors described below.

Cardiovascular Factors: Several groups of investigators have drawn attention to cardiovascular responses in ventilator-dependent patients and have emphasized the potential for ventilatory support reductions to induce ischemia or heart failure in susceptible patients with limited cardiac reserve.[9,89–93] Putative mechanisms include the following: (1) increased metabolic demand and hence circulatory demands that are associated with the transition from mechanical ventilation to spontaneous breathing in patients with limited cardiac reserve; (2) increases in venous return as the contracting diaphragm displaces blood from the abdomen to the thorax; and (3) the increased left ventricular afterload that is imposed by negative pleural pressure swings. Lemaire and colleagues demonstrated left ventricular dysfunction (ie, the pulmonary capillary wedge pressure increased from 8 to 25 mm Hg) during failed ventilator withdrawal attempts in 15 patients with COPD. Following diuresis, 9 of these 15 patients were successfully withdrawn from the ventilator.[90]

Psychological Factors: Psychological factors may be among the most important nonrespiratory factors leading to ventilator dependence. Fear of the loss of an apparent life support system as well as social/familial/economic issues all may play a role. Stress can be minimized by frequent communication among the staff, the patient, and the patient's family.[94] Environmental stimulation using television, radio, or books also appears to improve psychological functioning.[2] Ambulation using a portable ventilator (or bagging) has been shown to benefit attitudes and outlooks in long-term ventilator-dependent patients. Sleep deprivation may cause impairment of the respiratory control system,[95] although this may be related to accompanying factors rather than to sleep deprivation per se.[96] Finally, biofeedback may be helpful in decreasing the weaning time in patients who are having difficulty withdrawing from ventilatory support.[97–98]

Criteria to Assess Ventilator Dependence

Introduction

The process of discontinuing mechanical ventilatory support begins with a recognition of adequate recovery from acute respiratory failure. Thereafter, careful clinical assessments are required to determine the patient's readiness for subsequent discontinuation of ventilatory support and, ultimately, extubation. To facilitate this process, investigators have focused on identifying objective criteria to determine the answers to the following questions: When can efforts to discontinue ventilation be initiated? What assessment strategies will best identify the patient who is ready for ventilator discontinuation? When should extubation be carried out, and how can extubation outcome best be predicted?

Evidence to answer these questions comes largely from observational studies in which a certain parameter (or set of parameters) is compared in a group of patients who either successfully or unsuccessfully have been removed from the ventilator. The general goal of these studies is to find "predictors" of outcome. Evaluating the results from these types of studies can be difficult for several reasons.

First, the "aggressiveness" of the clinician/investigator's weaning and discontinuation philosophy needs to be understood, as it will affect the performance of a given predictor. A very aggressive clinical philosophy will maximize the number of patients withdrawn from ventilatory support but could also result in a number of premature discontinuations, with a subsequent need for reintubations and/or reinstitution of support. In contrast, a less aggressive clinical philosophy will minimize premature discontinuations but could also unnecessarily prolong ventilatory support in other patients. Unfortunately, there are no good data to help clinicians to determine the best balance between premature and delayed discontinuations in evaluating a given discontinuation strategy. Clearly, extubation failure should be avoided whenever possible because the need for reintubation carries an 8-fold higher odds ratio for nosocomial pneumonia[99] and a 6-fold to 12-fold increased mortality risk.[100–103] In contrast, the maintenance of unnecessary ventilator support carries its own burden of patient risk for infection and other complications.[104,105] Reported reintubation rates range from 4% to 23% for different intensive care unit (ICU) populations,[100,101,103,104,106–110] and may be as high as 33% in patients with mental status changes and neurological impairment.[103] Although the optimal rate of reintubation is not known, it would seem likely to rest between 5% and 15%.

Second, a number of methodological problems exist with most of these observational studies. For instance, patients are recruited into these studies because investigators believe that there is some reasonable chance of success for ventilator discontinuation. These "entry" criteria often include some form of clinical judgment or intuition, making results from one study difficult to compare to another. In addition, clinician/investigators deciding to proceed with ventilator discontinuation/extubation often have not been blinded to the parameters being analyzed as possible predictors. Indeed, the parameter being analyzed may often enter into the clinical decision on whether either to continue or to discontinue ventilatory support. Other methodological problems with these observational studies include different measurement techniques of a given parameter from study to study, large coefficients of variation with repeated measurements or from study to study of a given parameter,[111,112] different patient populations (eg, long-term vs short-term ventilator dependence),[113,114] and the absence of objective criteria to determine a patient's tolerance for a trial of either discontinuation or extubation.

Third, assessed outcomes differ from study to study. Some investigators have examined successful tolerance of a spontaneous breathing trial (SBT), others have used permanent discontinuation of the ventilator, and others have combined successful discontinuation and extubation. This latter approach is not optimal, given the differences in the pathophysiology of discontinuation versus extubation failure (see below).[102,106] In addition, different studies use different durations of ventilator discontinuation or extubation to define success or failure. Although 24–48 hours of unassisted breathing often is considered to define the successful discontinuation of ventilator support, many studies use shorter time periods to indicate success and often do not report subsequent reintubation rates or the need to reinstitute mechanical ventilatory support.

Fourth, a number of ways have been used to express predictor performance, and many can be confusing or misleading. Traditional indexes of diagnostic test power include sensitivity/specificity and positive/negative predictive values. These indexes are limited, however, in that they rely on a single cut point or threshold and that they do not provide an easy way to go from pretest likelihood or probability, through testing, to a posttest probability. The McMaster AHCPR report[4] recommends the use of likelihood ratios (LRs), and these will be used in this report to describe predictor performance. The LR is an expression of the odds that a given test result will be present in a patient with a given condition compared to a patient without the condition. An LR > 1 indicates that the probability of success increases, while values < 1 indicate that the probability of failure increases. LRs between 0.5 and 2 indicate that a weaning parameter is associated with only small, clinically unimportant changes in the posttest probability of success or failure. In contrast LRs from 2 to 5 and from 0.3 to 0.5 correlate with small but potentially important changes in probability, while ratios of 5 to 10 or 0.1 to 0.3 correlate with more clinically important changes

Table 3. Criteria Used in Weaning/Discontinuation Studies to Determine Whether Patients Receiving High Levels of Ventilatory Support Can Be Considered for Discontinuation (ie, Entered Into the Trials)*

Objective Measurements	Adequate oxygenation (eg, $P_{O_2} \geq 60$ mm Hg on $F_{IO_2} \leq 0.4$; PEEP $\leq$ 5–10 cm H_2O; $P_{O_2}/F_{IO_2} \geq 150$–300)
	Stable cardiovascular system (eg, HR $\leq$ 140 beats/min; stable blood pressure; no or minimal vasopressors)
	Afebrile (eg, temperature $< 38°C$)
	No significant respiratory acidosis
	Adequate hemoglobin (eg, Hgb $\geq$ 8–10 g/dL)
	Adequate mentation (eg, arousable, GCS $\geq$ 13, no continuous sedative infusions
	Stable metabolic status (eg, acceptable electrolytes)
Subjective Clinical Assessments	Resolution of disease acute phase; physician believes discontinuation possible; adequate cough

P_{O_2} = partial pressure of oxygen
F_{IO_2} = fraction of inspired oxygen
PEEP = positive end-expiratory pressure
HR = heart rate
GCS = Glasgow Coma Scale
*Adapted from References 101–103, 107–109, 119, and 120

in probability. Ratios of > 10 or < 0.1 correlate with very large changes in probability.[115]

Finally, because some investigators report data as continuous values (eg, means) rather than providing defined threshold values, combining studies using meta-analytic techniques often cannot be done.

Recommendation 2. *Patients receiving mechanical ventilation for respiratory failure should undergo a formal assessment of discontinuation potential if the following criteria are satisfied:*

1. Evidence for some reversal of the underlying cause of respiratory failure;

2. Adequate oxygenation (eg, $P_{aO2}/F_{IO_2} > 150$–200; requiring positive end-expiratory pressure [PEEP] $\leq$ 5–8 cm H_2O; $F_{IO_2} \leq 0.4$–0.5) and pH (eg, ≥ 7.25);

3. Hemodynamic stability as defined by the absence of active myocardial ischemia and the absence of clinically important hypotension (ie, a condition requiring no vasopressor therapy or therapy with only low-dose vasopressors such as dopamine or dobutamine < 5 μg/kg/min); and

4. The capability to initiate an inspiratory effort.

The decision to use these criteria must be individualized. Some patients not satisfying all of the above the criteria (eg, patients with chronic hypoxemia below the thresholds cited) may be ready for attempts at discontinuation of mechanical ventilation.

Rationale and Evidence (Grade B)

While some investigators argue that the process of discontinuation starts as soon as the patient is intubated, it would seem reasonable that an appropriate level of ventilatory support should be maintained until the underlying cause of acute respiratory failure and any complicating issues have shown some sign of reversal. Indeed, patients with unresolving respiratory failure who require high levels of ventilatory support are probably at high risk for respiratory muscle fatigue (and the consequent prolongation of the need for mechanical ventilation) if aggressive reductions in support are undertaken.[50,52,116–118]

The criteria used by clinicians to define disease "reversal," however, have been neither defined nor prospectively evaluated in a randomized controlled trial. Rather, various combinations of subjective assessment and objective criteria (eg, usually gas exchange improvement, mental status improvement, neuromuscular function assessments, and radiographic signs) that may serve as surrogate markers of recovery have been employed (Table 3).[101–103,107–109,119,120] It should be noted, however, that some patients who have not ever met one or more of these criteria still have been shown to be capable of eventual liberation from the ventilator.[104]

These "clinical assessments" of the status of the patient's respiratory failure, however, are not enough to make decisions on the discontinuation of support. For example, one survey[121] of intensivists using clinical judgment to assess the potential for discontinuation found a sensitivity of only 35% (6 of 17 patients who were successfully discontinued were identified) and a specificity of 79% (11 of 14 who failed discontinuation were identified). Moreover, in 2 large trials,[107,109] despite the presence of apparent disease stability/reversal prior to performing a screening SBT, the managing clinicians did not recognize that discontinuation was feasible in almost two thirds of the subjects. Thus, the conclusion is that some evidence of "clinical" stability/reversal is a key first step in assessing for discontinuation potential but that more focused assessments are needed before deciding to continue or discontinue ventilatory support.

Recommendation 3. *Formal discontinuation assessments for patients receiving mechanical ventilation for respiratory failure should be done during spontaneous breathing rather than while the patient is still receiving substantial ventilatory support. An initial brief period of spontaneous breathing can be used to assess the capability of continuing onto a formal SBT. The criteria with which to assess patient tolerance during SBTs are the respiratory pattern, adequacy of gas exchange, hemodynamic stability, and subjective comfort. The tolerance of SBTs lasting 30 to*

Table 4. Measurements Performed Either While Patient Was Receiving Ventilatory Support or During a Brief Period of Spontaneous Breathing That Have Been Shown to Have Statistically Significant LRs to Predict the Outcome of a Ventilator Discontinuation Effort in More Than One Study

Parameter	Studies (*n*)	Threshold Values	Positive LRs Range
Measured on Ventilator			
$\dot{V}_E$	20	10–15 L/min	0.81–2.37
NIF	10	−20 to −30 cm H_2O	0.23–2.45*
$P_{I_{max}}$	16	−15 to −30 cm H_2O	0.98–3.01
$P_{0.1}/P_{I_{max}}$	4	0.30	2.14–25.3
CROP score	2	13	1.05–19.74
Measured During a Brief Period of Spontaneous Breathing			
RR	24	30–38 breaths/min	1.00–3.89
V_T	18	325–408 mL (4–6 mL/kg)	0.71–3.83
f/V_T ratio	20	60–105/L	0.84–4.67

LR = likelihood ratio
$\dot{V}_E$ = minute ventilation
*1 study reported a likelihood ratio (LR) of 35.79.
NIF = negative inspiratory force (maximum inspiratory pressure)
$P_{I_{max}}$ = maximum inspiratory pressure
$P_{0.1}$ = mouth occlusion pressure 0.1 s after the onset of inspiratory effort
CROP = index including compliance, respiratory rate, oxygenation, and pressure
RR = respiratory rate
V_T = tidal volume
f/V_T = respiratory rate/tidal volume ratio

Table 5. Frequency of Tolerating an SBT in Selected Patients and Rate of Permanent Ventilator Discontinuation Following a Successful SBT

Study	Patients Receiving SBT	Patients Tolerating SBT *n* (%)	Patients Discontinuing Ventilation	Patients Having Ventilation Reinstituted *n* (%)
Esteban et al[105]	546	416 (76)	372	58 (16)
Ely et al[108]	113	88 (78)	65	5 (4)
Dojat et al[110]	38	22 (58)	22	5 (23)
Esteban et al[101]	246	192 (78)	192	36 (19)
Esteban et al[102]	270*	237 (89)	237	32 (14)
Esteban et al[102]	256†	216 (84)	216	29 (13)

SBT = spontaneous breathing trial
*30 min SBT
†120 min SBT

120 minutes should prompt consideration for permanent ventilator discontinuation.

Rationale and Evidence (Grade A)

Because clinical impression is so inaccurate in determining whether or not a patient meeting the criteria listed in Table 3 will successfully discontinue ventilatory support, a more focused assessment of discontinuation potential is necessary. These assessments can be performed either during spontaneous breathing or while the patient is still receiving substantial ventilatory support. These assessments can be used not only to drive decisions on weaning and discontinuation (ie, functioning as predictors) but also to offer insight into mechanisms of discontinuation failures.

The McMaster AHCPR report[4] found evidence in the literature supporting a possible role for 66 specific measurements as predictors. Some of these (eg, the negative effects of the duration of mechanical ventilation, and the length of/difficulty of surgery[44,122–124]) were derived from general clinical observations, but most were from studies on focused assessments of the patient's respiratory system. From these, the McMaster AHCPR group identified 8 parameters that had consistently significant LRs to predict successful discontinuation in several studies. Some of these measurements are made while the patient is still receiving ventilatory support; others require an assessment during a brief period of spontaneous breathing. These parameters, their threshold values, and the range of reported LRs are given in Table 4. It should be noted that despite the sta-

Table 6. Criteria Used in Several Large Trials* to Define Tolerance of an SBT

Objective measurements indicating tolerance/success	Gas exchange acceptability ($S_{pO_2} \geq$ 85–90%; $P_{O_2} \geq$ 50–60 mm Hg; pH $\geq$ 7.32; increase in $P_{aCO_2} \leq$ 10 mm Hg)
	Hemodynamic stability (HR < 120–140 beats/min; HR not changed > 20%; systolic BP < 180–200 mm Hg and > 90 mm Hg; BP not changed > 20%, no pressors required)
	Stable ventilatory pattern (eg, RR $\leq$ 30–35 breaths/min, RR not changed > 50%)
Subjective clinical assessments indicating intolerance/failure	Change in mental status (eg, somnolence, coma, agitation, anxiety)
	Onset or worsening of discomfort
	Diaphoresis
	Signs of increased work of breathing (use of accessory respiratory muscles, thoracoabdominal paradox)

SBT = spontaneous breathing trial
S_{pO_2} = oxygen saturation measured by pulse oximetry
P_{O_2} = partial pressure of oxygen
P_{aCO_2} = arterial partial pressure of carbon dioxide
HR = heart rate
RR = respiratory rate
*References 101, 102, 105, 108–110, 119, and 120.

tistical significance of these parameters, the generally low LRs indicate that the clinical applicability of these parameters alone to individual patients is low.

Although assessments that are performed while a patient is receiving substantial ventilatory support or during a brief period of spontaneous breathing (Tables 3 and 4) can yield important information about discontinuation potential, assessments that are performed during a formal, carefully monitored SBT appear to provide the most useful information to guide clinical decision-making regarding discontinuation. Indeed, because of the efficacy and safety of a properly monitored SBT (see below), the assessments in Table 4 that are performed to predict SBT outcome are generally unnecessary.

In concept, the SBT should be expected to perform well, as it is the most direct way to assess a patient's performance without ventilatory support. Indeed, the evidence for this concept is quite strong. As can be seen in Table 5, multiple studies have found that patients tolerant of SBTs that are 30 to 120 min in length were found to have successful discontinuations at least 77% of the time.

Because the 12 to 42% of patients in the Table 5 studies failing the SBT were not systematically removed from ventilatory support, the ability of a failed SBT to predict the need for ventilator dependence (ie, negative predictive value) cannot be formally assessed. Indeed, it is conceivable that iatrogenic factors such as endotracheal tube discomfort or continuous positive airway pressure (CPAP) demand-valve insensitivity/unresponsiveness, rather than true ventilator dependence, caused the failure of the SBT in at least some of these patients.[125] Thus, it is thus unclear how many patients who are unable to tolerate an SBT would still be able to tolerate long-term ventilator discontinuation. Although the number is likely to be small, it is probably not zero, and this needs to be considered when dealing with patients who repeatedly fail an SBT.

The criteria used to define SBT "tolerance" are often integrated indexes since, as noted above, single parameters alone perform so poorly. These integrated indexes usually include several physiologic parameters as well as clinical judgment, incorporating such difficult-to-quantify factors as "anxiety," "discomfort," and "clinical appearance." The criteria that have been used in several large trials are given in Table 6.

A potential concern about the SBT is safety. Although unnecessary prolongation of a failing SBT conceivably could precipitate muscle fatigue, hemodynamic instability, discomfort, or worsened gas exchange,[19,50,117,126,127] there are no data showing that SBTs contribute to any adverse outcomes if terminated promptly when failure is recognized. Indeed, in a cohort of > 1,000 patients in whom SBTs were routinely administered and properly monitored as part of a protocol, only one adverse event was thought to be even possibly associated with the SBT.[104]

There is evidence that the detrimental effects of ventilatory muscle overload, if it is going to occur, often occurs early in the SBT.[73,108,110,128] Thus, the initial few minutes of an SBT should be monitored closely, before a decision is made to continue (this is often referred to as the "screening" phase of an SBT). Thereafter, the patient should continue the trial for at least 30 min, but for not > 120 min,[102] to assure maximal sensitivity and safety. It also appears that whether the SBT is performed with low levels of CPAP (eg, 5 cm H_2O), low levels of pressure support (eg, 5–7 cm H_2O), or simply as "T-piece" breathing has little effect on outcome.[101,129–131] CPAP, however, conceivably could enhance breath triggering in patients with significant auto-PEEP.[132,133]

Recommendation 4. *The removal of the artificial airway from a patient who has successfully been discontinued from ventilatory support should be based on assessments of airway patency and the ability of the patient to protect the airway.*

Rationale and Evidence (Grade C)

Extubation failure can occur for reasons distinct from those that cause discontinuation failure. Examples include upper airway obstruction or inability to protect the airway

and clear secretions. The risk of postextubation upper airway obstruction increases with the duration of mechanical ventilation, female gender, trauma, and repeated or traumatic intubation.[106] The detection of an air leak during mechanical ventilation when the endotracheal tube balloon is deflated can be used to assess the patency of the upper airway (cuff leak test).[134] In a study of medical patients,[135] a cuff leak < 110 mL (ie, average of 3 values on 6 consecutive breaths) measured during assist control ventilation within 24 hours of extubation identified patients at high risk for postextubation stridor. Although others have not confirmed the utility of the cuff leak test for predicting postextubation stridor,[136] many patients who develop this can be treated with steroids and/or epinephrine (and possibly with noninvasive ventilation and/or heliox) and do not necessarily need to be reintubated. Steroids and/or epinephrine could also be used 24 hours prior to extubation in patients with low cuff leak values. It is also important to note that a low value for cuff leak may actually be due to encrusted secretions around the tube rather than to a narrowed upper airway. Despite this, reintubation equipment (including tracheostomy equipment) should be readily available when extubating patients with a low cuff leak values.

The capacity to protect the airway and to expel secretions with an effective cough would seem to be vital for extubation success, although specific data supporting this concept are few. Successful extubations have been reported[137] in a select group of brain-injured comatose patients who were judged to be capable of protecting their airways. However, it is difficult to extrapolate this experience to more typical ICU patients, and many would argue that some capability of the patient to interact with the care team should be present before the removal of an artificial airway. Airway assessments generally include noting the quality of cough with airway suctioning, the absence of "excessive" secretions, or the frequency of airway suctioning (eg, every 2 h or more).[34,108,138] Coplin et al[137] devised an "airway care score," which semiquantitatively assesses cough, gag, suctioning frequency, and sputum quantity, viscosity, and character that predicted extubation outcome. Peak cough flows of > 160 L/min predict successful translaryngeal extubation or tracheostomy tube decannulation in neuromuscular- or spinal cord-injured patients.[139]

Managing the Patient Who Has Failed a Spontaneous Breathing Test

Introduction

The failure of a patient to complete an SBT raises 2 important questions. First, what caused the SBT failure, and are there readily reversible factors that can be corrected? Second, how should subsequent mechanical ventilatory support be managed? Specifically, should an SBT be tried again? If so, when? What form of ventilatory support should be provided in between SBTs and should support be at a constant high level or should efforts be made to routinely reduce the level of support gradually (ie, to wean support)?

Evaluating evidence addressing mechanical ventilatory support strategies is particularly problematic. This is because trials comparing 2 or more approaches to ventilator management compare not only the modes of ventilation but also how those modes are used. Ideally, trial design should be such that management philosophies and the aggressiveness of support reduction are similar in each strategy being evaluated. Unfortunately, this is often not the case, as investigator experience with one approach has a tendency to result in more favorable "rules" of support reduction for that approach compared to others.

Recommendation 5. *Patients receiving mechanical ventilation for respiratory failure who fail an SBT should have the cause for the failed SBT determined. Once reversible causes for failure are corrected, and if the patient still meets the criteria listed in Table 3, subsequent SBTs should be performed every 24 hours.*

Rationale and Evidence (Grade A)

Although failed SBTs are often a reflection of persistent respiratory system abnormalities,[52] a failed SBT should prompt a search for other causes or complicating factors (see the "Pathophysiology of Ventilator Dependence" section). Specific issues include the adequacy of pain control, the appropriateness of sedation, fluid status, bronchodilator needs, the control of myocardial ischemia, and the presence of other disease processes that either can be readily addressed or else can be considered when deciding to proceed further with discontinuation attempts.

Assuming medical management is optimized and that the patient who has failed an SBT still meets the criteria listed in Table 3, the following 2 questions involving subsequent SBTs arise: First, should SBTs be attempted again or should another approach to ventilator withdrawal be attempted? Second, if an SBT is attempted again, when should that be?

There are some data on which to base an answer to the first question. The one large randomized trial[107] that compared routine SBTs to 2 other weaning strategies that did not include SBTs provides compelling evidence that SBTs administered at least once daily shorten the discontinuation period compared to strategies that do not include daily SBTs. In addition, 2 studies[108,119] showing the success of protocol-driven ventilator discontinuation strategies over "usual care" both included daily SBTs. The subsequent use of routine subsequent SBTs in this patient population thus seems appropriate.

There are several lines of evidence that support waiting 24 hours before attempting an SBT again in these patients. First, except in patients recovering from anesthesia, muscle relaxants, and sedatives, respiratory system abnormalities rarely recover over a short period of hours and thus frequent SBTs over a day may not be expected to be helpful. Supporting this are data from Jubran and Tobin[52] showing that failed SBTs often are due to persistent respiratory system mechanical abnormalities that are unlikely to reverse rapidly. Second, there are data suggesting that a failed SBT may result in some degree of respiratory muscle fatigue.[50,117,118] If so, studies[126,140] conducted in healthy subjects suggest that recovery may not be complete for anywhere from several hours to > 24 hours. Third, the trial by Esteban et al[107] specifically addressed this issue and provided strong evidence that twice-daily SBTs offer no advantage over a single SBT and, thus, would serve only to consume unnecessary clinical resources.

Recommendation 6. *Patients receiving mechanical ventilation for respiratory failure who fail an SBT should receive a stable, nonfatiguing, comfortable form of ventilatory support.*

Rationale and Evidence (Grade B)

There are a number of ventilator modes that can provide substantial ventilatory support as well as the means to reduce partial ventilatory support in patients who have failed an SBT (Table 7). A key question, however, is whether attempts at gradually lowering the level of support (weaning) offer advantages over a more stable, unchanging level of support between SBTs. The arguments for using gradual reductions are (1) that muscle conditioning might occur if ventilatory loads are placed on the patient's muscles and (2) that the transition to extubation or to an SBT might be easier from a low level of support than from a high level of support. Data supporting either of these claims, however, are few. However, maintaining a stable level of support between SBTs reduces the risk of precipitating ventilatory muscle overload from overly aggressive support reduction. It also offers a substantial resource consumption advantage in that it requires far less practitioner time. The study by Esteban et al[107] partially addressed this issue in that it compared daily SBTs (and a stable level of support in those who failed) to 2 other approaches using gradual reductions in support (ie, weaning with pressure support or intermittent mandatory ventilation [IMV]) and demonstrated that the daily SBT with stable support between tests permitted the most rapid discontinuation. What has not been addressed, however, is whether gradual support reductions *coupled* with daily SBTs offer any advantages.

The McMaster AHCPR report[4] identified 3 other randomized trials[109,141,142] that compared gradual reduction strategies using different modes but *not* routine daily SBTs. The study by Brochard et al[109] was the most similar in design to the study by Esteban et al[107] that was noted before, and it included a pressure-support group and an IMV group. A third group received gradually increasing fixed periods of spontaneous breathing that were designed only to provide brief periods of work and not specifically to test for discontinuation (ie, they were not routine daily SBTs, as defined above). The results showed that their use of gradually lengthening spontaneous breathing periods was inferior to other strategies and, like the Esteban trial, the pressure support strategy was easier to reduce than the IMV strategy. The other 2 randomized trials[141,142] that were identified by the McMaster AHCPR report were much smaller than the Esteban et al[107] and Brochard et al,[109] and both suggested that pressure support was easier to reduce than IMV alone. Because none of these studies offer evidence that gradual support strategies are superior to stable support strategies between SBTs, the clinical focus for the 24 hours after a failed SBT should be on maintaining adequate muscle unloading, optimizing comfort (and thus sedation needs), and avoiding complications, rather than aggressive ventilatory support reduction.

Ventilator modes and settings can affect these goals.[143] Assisted modes of ventilation (as opposed to machine-controlled modes) are generally preferable in this setting

Table 7. Modes of Partial Ventilator Support

Mode	Patient Work Adjusted By
SIMV	Number of machine breaths supplied (ie, the fewer the number of machine breaths, the more spontaneous breaths are required)
PSV	Level of inspiratory pressure assistance with spontaneous efforts
SIMV + PSV	Combining the adjustments of SIMV and PSV
VS	PSV with a "guaranteed" minimum V_T (PSV level adjusts automatically according to clinician V_T setting)
VAPS(PA)	PSV with "guaranteed" minimum V_T (additional flow is supplied at end inspiration if necessary to provide clinician V_T setting)
MMV	SIMV with a "guaranteed" $\dot{V}_E$ (machine breath rate automatically adjusts according to clinician $\dot{V}_E$ setting)
APRV	Pressure difference between inflation and release (ie, the less the pressure difference, the more spontaneous breaths are required)

SIMV = synchronized intermittent mandatory ventilation
PSV = pressure support ventilation
VS = volume support
VAPS(PA) = volume-assured pressure support (pressure augmentation)
V_T = tidal volume
MMV = mandatory minute ventilation
$\dot{V}_E$ = minute ventilation
APRV = airway pressure-release ventilation

because they allow patient muscle activity and some patient control over the ventilatory pattern. Although good clinical supporting data are lacking, these features may help to avoid muscle disuse atrophy[24] and may reduce sedation needs in these types of patients.[143] With assisted modes, sensitive/responsive ventilator triggering systems,[144–147] applied PEEP in the presence of a triggering threshold load from auto-PEEP,[132,133] flow patterns matched to patient demand,[148–152] and appropriate ventilator cycling to avoid air trapping[153,154] are all important to consider in achieving patient comfort and minimizing imposed loads.

In recent years, several ventilator support modes (volume support,[155] adaptive support ventilation (ASV),[155,156] minimum minute ventilation (MMV),[155] and a knowledge-based system for adjusting pressure support[110,157]) have been developed in an attempt to "automatically" wean patients by feedback from one or more ventilator-measured parameters. Minimum minute ventilation set at either 75% of measured minute ventilation[158] or to a carbon dioxide target,[120] and a knowledge-based system for adjusting pressure support[110,157] all have been shown to be capable of automatically reducing support safely in selected populations. However, none of these has been compared to the daily SBT approach described above. Moreover, the premises underlying some of these feedback features (eg, that an ideal volume can be set for volume support or that an ideal ventilatory pattern based on respiratory system mechanics can be set for ASV) may be flawed, especially in sick patients. Indeed, potentially flawed feedback logic may, in fact, delay support reduction. Further work is clearly needed to establish the role (if any) of these automated approaches.

There has been increasing interest in the use of noninvasive positive-pressure ventilation (NPPV) in recent years. Although NPPV has been used primarily as a method to avoid intubation, it has also been used as a technique to facilitate the discontinuation of invasive ventilatory support. Data from the pooling of results of 2 prospective, randomized controlled trials[159,160] in patients with chronic respiratory disease suggest the need for reductions in the durations of mechanical ventilation, ICU stay, mortality, and the incidence of nosocomial pneumonia with postextubation support provided by NPPV. Appropriate patient selection and the feasibility of the widespread application of these findings remains to be determined.

Recommendation 7. *Anesthesia/sedation strategies and ventilator management aimed at early extubation should be used in postsurgical patients.*

Rationale and Evidence (Grade A)

The postsurgical patient poses unique problems for ventilator discontinuation. In these patients, depressed respiratory drive and pain issues are the major reasons for ventilator dependence. Optimal sedation, pain management, and ventilator strategies offer opportunities to shorten the duration of mechanical ventilation.

The McMaster AHCPR report[4] identified 5 randomized controlled trials in postcardiac surgery patients[161–165] that demonstrated that a lower anesthetic/sedation regimen permitted earlier extubation. The pooled results showed a mean effect of 7 hours. Similar effects were found using these approaches in other postsurgical populations.[166–170]

Ventilator modes that guarantee a certain breath rate and minute ventilation (assist control modes, IMV, and MMV) are important in patients with unreliable respiratory drives. However, frequent assessments and support reductions are necessary since recovery in these patients usually occurs over only a few hours. Aggressive support reduction strategies have been shown to lead to earlier discontinuations of ventilation.[166,169] Conceptually, the immediate postoperative patient might be ideally suited for simple automatic feedback modes that provide a backup form of support (eg, MMV or ASV).[120,156,158] Data showing improved outcomes or lower costs with these automated approaches, however, are lacking.

Recommendation 8. *Weaning/discontinuation protocols designed for nonphysician health care professionals (HCPs) should be developed and implemented by ICUs. Protocols aimed at optimizing sedation should also be developed and implemented.*

Rationale and Evidence (Grade A)

There is clear evidence that nonphysician HCPs (eg, respiratory therapists and nurses) can execute protocols that enhance clinical outcomes and reduce costs for critically ill patients.[171] In recent years, 3 randomized controlled trials incorporating 1,042 patients also have demonstrated that outcomes for mechanically ventilated patients who were managed using HCP-driven protocols were improved over those of control patients managed with standard care. Specifically, Ely et al[108] published the results of a 2-step protocol driven by HCPs using a daily screening procedure followed by an SBT in those who met screening criteria. The discontinuation of mechanical ventilation was then recommended for patients tolerating the SBT. Although the 151 patients managed with the protocol had a higher severity of illness than the 149 control subjects, they were removed from the ventilator 1.5 days earlier (with 2 days less weaning), had 50% fewer complications related to the ventilator, and had mean ICU costs of care that were lower by > $5,000 per patient. In a slightly larger trial with a more diverse patient population, Kollef et al[119] used 3 different nonphysician-HCP-driven protocols and showed that the mean duration of mechanical

ventilation could be reduced by 30 hours. Finally, Marelich et al[172] showed that the duration of mechanical ventilatory support could be reduced almost 50% using nurse-driven and therapist-driven protocols ($p = 0.0001$).

The reproducibility of benefit for using various protocols in different ICUs and institutions suggests that it is the use of a standardized approach to management rather than any specific modality of ventilator support that improves outcomes. Indeed, when other key features in the management of mechanically ventilated patients, such as sedation and analgesia, also are subjected to protocols, further reductions in the time spent receiving mechanical ventilation can be achieved. For example, in a randomized controlled trial[173] of a nursing-implemented sedation protocol for 321 patients receiving mechanical ventilation, the use of the protocol was associated with a 50% reduction in the duration of mechanical ventilation and 2-day and 3-day reductions in the median ICU and lengths of hospital stay, respectively (all p values < 0.01). More recently, Kress et al[174] published the results of a randomized controlled trial of 128 patients showing that a daily spontaneous awakening trial was associated with 2 days fewer spent receiving mechanical ventilation ($p = 0.004$) and a 3-day shorter ICU stay ($p = 0.02$).

The data do not support endorsing any one ventilator discontinuation protocol, and the choice of a specific protocol is best left to the individual institution. In designing these protocols, consideration should be given to other recommendations in this document as well as to the specific patient populations. For instance, medical patients with severe lung injury might benefit from one type of management strategy (see recommendations 2 to 5), whereas surgical patients recovering from anesthesia might benefit from another strategy (see recommendation 7). In the context of emerging data about the benefits of NPPV[159,160] and the substantial roles of HCPs in providing this treatment, there should be efforts made to develop HCP-driven protocols for this modality.

While each institution must customize the protocols to local practice, there are important general concepts that may ease the process of implementation and enhance success.

First, protocols should not be used to replace clinical judgment, but rather to complement it. Protocols are meant as guides and can serve as the general default management decision unless the managing clinician can justify a departure from the protocol. Any such departure should be carefully assessed and used to guide possible future modifications of the protocol.

Second, protocols should not be viewed as static constructs, but rather as dynamic tools in evolution, which can be modified to accommodate new data and/or clinical practice patterns. More studies regarding the impact of protocol-based ventilator management are needed for specific patient populations (eg, neurosurgical patients[175] and trauma patients[176]), in specific organizational structures (eg, open vs closed units and teaching vs community hospitals), and using computer-assisted decision-making.

Third, institutions must be prepared to commit the necessary resources to develop and implement protocols.[177] For instance, the effective implementation of protocols requires adequate staffing, as it has been shown that if staffing is reduced below certain thresholds, clinical outcomes may be jeopardized.[178,179] Indeed, in the specific context of the discontinuation of mechanical ventilation, reductions in nurse/patient ratios have been associated with prolonged duration of mechanical ventilation.[180]

Role of Tracheotomy in Ventilator-Dependent Patients

Introduction

Tracheotomy is commonly performed for critically ill, ventilator-dependent patients to provide long-term airway access. The benefits commonly ascribed to tracheotomy, compared to prolonged translaryngeal intubation, include improved patient comfort, more effective airway suctioning, decreased airway resistance, enhanced patient mobility, increased opportunities for articulated speech, ability to eat orally, and a more secure airway. Conceptually, these advantages might result in fewer ventilator complications (eg, ventilator-associated pneumonia), accelerated weaning from mechanical ventilation, and the ability to transfer ventilator-dependent patients from the ICU. Concern, however, exists about the risks associated with the procedure and the costs involved.

The impact of tracheotomy on the duration of mechanical ventilation and ICU outcomes in general has been examined by several different study designs—none of them ideal. Most studies are retrospective, although a few prospective studies have been performed. A serious problem is that many studies assigned patients to treatment groups on the basis of physician practice patterns rather than random assignment. Those studies that used random assignment frequently used quasi-randomization methods (eg, every other patient, every other day, hospital record number, odd-even days). Studies have compared patients undergoing tracheotomy versus those not undergoing tracheotomy and patients undergoing early tracheotomy versus those undergoing late tracheotomy. The definition of early versus late tracheotomy varies between studies. "Early" may be defined as a period as short as 2 days to as late as 10 days after the start.

Patient populations included in studies also vary widely between investigations and include general surgical and medical patients in some studies and specific patient groups

(eg, trauma patients or head-injured patients) in other studies. Most studies have design flaws in the collection and analysis of data, foremost of which is the absence of blinding. The absence of blinding is especially important considering that no study has used explicit, systematic protocols for weaning to control for any effects of tracheotomy on altering the approaches of clinicians to weaning. Finally, an outcome such as transfer to a non-ICU setting may depend on local resources, such as the availability of a non-ICU ventilator service.

Because there is such a surprisingly small amount of quality data regarding the relative impact of tracheotomy in terms of patient outcome relative to prolonged translaryngeal intubation, past recommendations for timing the procedure to achieve these benefits have been based on expert consensus.[181]

Recommendation 9. *Tracheotomy should be considered after an initial period of stabilization on the ventilator when it becomes apparent that the patient will require prolonged ventilator assistance. Tracheotomy should then be performed when the patient appears likely to gain one or more of the benefits ascribed to the procedure. Patients who may derive particular benefit from early tracheotomy are the following:*

- *Those requiring high levels of sedation to tolerate translaryngeal tubes*
- *Those with marginal respiratory mechanics (often manifested as tachypnea) in whom a tracheostomy tube having lower resistance might reduce the risk of muscle overload*
- *Those who may derive psychological benefit from the ability to eat orally, communicate by articulated speech, and experience enhanced mobility; and*
- *Those in whom enhanced mobility may assist physical therapy efforts*

Rationale and Evidence (Grade B)

While carrying some risks, tracheotomies in ventilator-dependent patients are generally safe. The problems associated with tracheotomy include perioperative complications related to the surgery, long-term airway injury, and the cost of the procedure.

Patient series reported during the early 1980s[182] suggested that tracheotomy had a high risk of perioperative and long-term airway complications such as tracheal stenosis. More recent studies,[183–185] however, have established that standard surgical tracheotomy can be performed with an acceptably low risk of perioperative complications. Regarding long-term risks, analyses of longitudinal studies suggest that the risk of tracheal stenosis after tracheotomy is not clearly higher than the risks of subglottic stenosis from prolonged translaryngeal intubation.[186] Also, the nonrandomized studies commonly reported in the literature bias results toward greater long-term airway injury in patients undergoing tracheotomy because the procedure was performed after a prolonged period of translaryngeal intubation, which may prime the airway for damage from a subsequent tracheotomy.[182,187–189] Finally, the cost of tracheotomy can be lowered if it is performed in the ICU rather than an operating room, either by the standard surgical or percutaneous dilational technique.[186,187] Even when tracheotomy is performed in an operating room, the cost may be balanced by cost savings if a ventilator-dependent patient can be moved from an ICU setting after the placement of a tracheostomy. The actual cost benefits of tracheotomy, however, have not been established because no rigorous cost-effectiveness analyses have been performed.

Given the above conditions, it seems reasonable to conclude that none of the potential problems with tracheotomy is of sufficient magnitude to make tracheotomy any less clinically acceptable compared with other procedures commonly performed in critically ill patients.

Potentially, the most important beneficial outcome from a tracheotomy would be to facilitate the discontinuation of mechanical ventilatory support. Supporting evidence comes both from observations on "intermediate" end points (eg, comfort and mobility, decreased airway resistance, and a lower incidence of ventilator-associated pneumonia) as well as ICU outcome studies examining the duration of mechanical ventilation, ICU length of stay (LOS), and mortality. This evidence is reviewed below.

Improved Patient Comfort: No prospective outcome studies in general populations of ventilator-dependent patients using validated measurement tools have established that tracheotomy results in greater patient comfort or mobility, compared with prolonged translaryngeal intubation. Indeed, to our knowledge, only one study[183] has attempted to document this by reporting that interviewed ICU caregivers believed ventilated patients were more comfortable after tracheotomy. Despite this lack of data, the general clinical consensus is that patients supported with long-term mechanical ventilation have less facial discomfort when nasotracheal or orotracheal endotracheal tubes are removed and a tracheotomy is performed. Furthermore, patient well-being is thought to be promoted by a tracheotomy through its effects on assisting articulated speech, oral nutrition, and mobility, which may promote the discontinuation of sedatives and analgesics. The maintenance of continuous sedation has been associated with the prolongation of mechanical ventilation,[190] but the effects of tracheotomy on sedation usage have not been studied specifically.

Table 8. The Impact of Tracheotomy on Mechanical Ventilation Outcome

Study	Patient Type (*n*)	Design	Outcome
Lesnik et al[201]	Trauma/101	Randomized early (6 d) vs late (21 d) tracheotomy	Vent days shorter with earlier tracheotomy
Blot et al[202]	Neutropenic/53	Retrospective early (2 d) vs late (> 7 d) tracheotomy or no tracheotomy	LOS longer in patients undergoing early tracheotomy
Koh et al[203]	Neurosurgical/49	Retrospective elective (9 d) vs failed extubation tracheotomy	LOS shorter in elective tracheotomy
Dunham and LaMonica[199]	Trauma/74	Prospective early (4 d) vs late (14 d) or no tracheotomy	No effect
El Nagger et al[204]	General acute respiratory failure/52	Prospective (3 d) vs delayed (> 10 d) tracheotomy	More patients weaned in delayed tracheotomy
Rodriguez et al[198]	Trauma/106	Randomized early (≤ 7 d) vs late (> 7 d) tracheotomy	LOS shorter in patients undergoing early tracheotomy but those weaned before late tracheotomy not considered
Sugerman et al[200]	Trauma/126	Randomized early (3–5 d) vs late (10–21 d) tracheotomy	No effect

LOS = length of stay

Decreasing Airway Resistance: Although the small radius of curvature of tracheostomy tubes increases turbulent air flow and airway resistance, the short length of tracheostomy tubes results in an overall lowering of airway resistance (and thus reduced patient muscle loading) when compared to standard endotracheal tubes in both laboratory and clinical settings[191–197] While the development of secretions will increase resistance in both tracheostomy and endotracheal tubes, easier suctioning and removable inner cannulas may reduce this effect in tracheostomy tubes.[195] The existing data thus indicate that airway resistance and muscle loading may decrease in some patients after the performance of tracheotomy, but the clinical impact of this improvement in pulmonary mechanics on weaning has not been established. Conceivably, patients with borderline pulmonary mechanics may benefit from tracheotomy because of decreased airway resistance, which becomes more clinically important with high respiratory rates.

Impact of Tracheotomy on Ventilator-Associated Pneumonia: Early tracheotomy and, alternatively, the avoidance of tracheotomy by maintaining a translaryngeal endotracheal tube in place both have been proposed as strategies to promote the successful discontinuation of mechanical ventilation by avoiding ventilator-associated pneumonia. Few data support the conclusion, however, that the timing of tracheotomy alters the risk of pneumonia. Three prospective studies have evaluated the relative risk of pneumonia in patients randomized to early versus late tracheotomy.[198–200] These studies examined 289 patients and found a relative risk for pneumonia (early tracheotomy vs late tracheotomy group) of only 0.88 (95% confidence interval 0.70 to 1.10). Considerable methodological flaws in these studies, however, do not allow firm conclusions to be drawn regarding the effects of tracheotomy on pneumonia risk. Presently, no data support the competing contentions that early tracheotomy either decreases or increases the risk of ventilator-associated pneumonia.

Outcome Studies: Impact of Tracheotomy on Duration of Mechanical Ventilation: The results of a number of studies examining ICU outcome (ie, ventilator days, ICU LOS, mortality) have been reported, and are summarized in Table 8.[198–204] Several of these studies were appraised in a systematic review.[205] The authors of this review concluded that insufficient evidence existed to support the contention that the timing of tracheotomy alters the duration of mechanical ventilation in critically ill patients. Also, the review identified multiple flaws in the available studies. There appears to be a clinical impression that timing of tracheotomy promotes discontinuation of mechanical ventilation in some ventilator-dependent patients, but not all. However, the quality of existing studies does little to support this clinical impression.

In the future, because of the difficulty in blinding caregivers to the presence or absence of tracheotomy, studies should use explicit weaning protocols to control for different levels of approaches toward weaning that the presence of a tracheotomy may invoke. Studies also could be improved by more rigorous patient inclusion and exclusion criteria, better accounting for dropouts, the use of conventional randomization methods, multicenter designs to allow sufficient sample sizes to determine the interaction of underlying conditions, and multivariate analysis techniques. Cost-effectiveness analysis

Table 9. Comparison of Observational Studies*

Variable	Indihar[213]	Latriano et al[214]	Petrak et al[215]	Clark and Theiss[216]	Bagley and Cooney[217]	Scheinhorn et al[208]	Carson et al[218]	Dasgupta et al[219]	Gracey et al[220]
Type of unit	NRCU	NRCU	RWC	RWC	RWC	RWC	RWC	NRCU	NRCU
Patients, No.	171	224	388	113	278	1,123	133	212	420
Age (y)†		67	72‡	65	67	69	71	68	67§
Sex (% women)	54	47	53	56	53	57	52	55	52§
Diagnoses precipitating ventilator dependency	COPD	medical > surgical	medical > surgical	medical = surgical	medical > surgical	medical > surgical	medical > surgical	medical = surgical	surgical > medical
ICU days† receiving ventilation	55	23	42	–	–	44	25	25‡	37§
Days† to weaning in ICU	39	43	–	–	43	39	–	13‡	10
% Weaned‖	34	51	51	47	38	56	38	60	60
% Survival to discharge	60	50	66	61	53	71	50	82	94
% Survival 12 mo after discontinuing ventilation	–	–	–	–	–	38	23	–	53¶

*Each with > 100 patients transferred for weaning from prolonged mechanical ventilation (> 21 prior ICU ventilator days).
NRCU = noninvasive respiratory care unit
RWC = regional weaning center
†Mean
‡Median
§Data from an earlier report on the same cohort of patients.[211]
‖Authors' data were recalculated so that "% weaned" signifies number weaned/number of patients who were admitted to the hospital ventilator-dependent
¶Value attained 4 years after discharge
Adapted from Reference 224

also would assist the determination of the value of tracheotomy for weaning.

The Role of Long-Term Facilities

Introduction

The patient who remains ventilator-dependent despite maximal medical/surgical therapy and aggressive attempts to remove ventilator support is becoming an increasing challenge for critical care practitioners. In 2 studies,[206,207] up to 20% of medical ICU patients met the 21-day Health Care Financing Administration definition of prolonged mechanical ventilation (PMV).[208] Better treatment and technology, no doubt, are playing a major role in the conversion of patients who would have died a decade ago into patients who survive today, but who have substantial remaining respiratory dysfunction.

Prior to the 1980s, these patients simply remained in ICUs and were managed using acute-care principles. The only other option was permanent ventilatory support in either the patient's home or in a nursing home. Financial pressures, coupled with the concept that the aggressive ICU mindset might not be optimal for the more slowly recovering patient, have led to creation of weaning facilities (both free-standing facilities and units within hospitals) that are potentially more cost-effective and better suited to meet the needs of these patients. A body of literature now is emerging that suggests that many patients who previously would have been deemed "unweanable" may achieve ventilator independence in such facilities.

Recommendation 10. *Unless there is evidence for clearly irreversible disease (eg, high spinal cord injury or advanced amyotrophic lateral sclerosis), a patient requiring prolonged mechanical ventilatory support for respiratory failure should not be considered permanently ventilator-dependent until 3 months of weaning attempts have failed.*

Rationale and Evidence (Grade B)

A critical clinical issue is determining whether a patient requiring PMV has any hope of ventilator discontinuation or whether he/she is to have lifelong ventilator dependence. Patients in the former category clearly need attempts at ventilator discontinuation to be pursued, whereas patients in the latter category are only being subjected to unnecessary episodes of worsening respiratory failure with such attempts. These latter patients, instead, need to have the clinical focus changed to establish a lifelong support program.

Data from a number of centers caring for the patient requiring long-term ventilation offer insight into this question. In the Barlow Regional Weaning Center experience,[208–210] patients with prolonged ventilator dependence following acute cardiorespiratory failure were still undergoing ventilator discontinuation up to 3 months (and, on occasion, 6 months) postintubation. Other studies[211,212] suggest similar results in postsurgical and medical populations. Data from these stud-

Table 10. Post-ICU Weaning Strategies

Study	Strategies to Decrease Ventilator Support to Approximately Half Full Support	Strategies to Increase SBT
Gracey et al[220]*	IMV/PSV	TTO
Scheinhorn et al[208]	IMV/PSV	VTM
Bagley and Cooney[217]	–	VTM/T-piece
Petrak et al[215]*	AC or PSV	VTM
Clark and Theiss[216]	PSV	VTM/T-piece
Latriano et al[214]	IMV/PSV	VTM/T-piece

ICU = intensive care unit
SBT = spontaneous breathing trial
*Data from personal communication
IMV = intermittent mandatory ventilation
PSV = pressure-support ventilation
TTO = transtracheal oxygen
VTM = Venti-Trach mask (tracheal collar)
AC = assist-control

ies on the time that patients spend, on average, dependent on ventilator support in the ICU (36 d) and during subsequent weaning in the post-ICU setting (31 d), suggest a time frame for the reasonable continuance of ventilator discontinuation attempts. Thus the weight of evidence is that several months of attempts at ventilator discontinuation are required before most patients who are receiving mechanical ventilation for acute respiratory failure can be declared to be permanently ventilator-dependent.

Recommendation 11. *Critical-care practitioners should familiarize themselves with facilities in their communities, or units in hospitals they staff, that specialize in managing patients who require prolonged dependence on mechanical ventilation. Such familiarization should include reviewing published peer-reviewed data from those units, if available. When medically stable for transfer, patients who have failed ventilator discontinuation attempts in the ICU should be transferred to those facilities that have demonstrated success and safety in accomplishing ventilator discontinuation.*

Rationale and Evidence (Grade C)

There are >30 studies on post-ICU weaning from PMV. Most of these studies are observational, a reflection of the inherent logistical difficulties in designing randomized controlled trials in such a heterogeneous population that is being treated in such diverse settings. Table 9 summarizes the studies that report outcomes in > 100 patients in which PMV is defined as > 21 days of ventilator dependency. Most of these studies[208,211–219] support the conclusion that ICU patients receiving PMV can be discontinued effectively and safely from ventilation when they are transferred to units dedicated to that activity. Efficacy is suggested in the fact that the data in Table 9 encompass 3,062 post-ICU patients and show that 1,588 (52%) successfully discontinued mechanical ventilation. Safety is suggested in that a 69% overall survival-to-discharge rate in this "chronically critically ill" cohort seems quite reasonable.

The facilities generating the data of Table 9 are of 2 basic types: (1) Most facilities, but not all, are licensed as long-term acute-care hospitals, which are required by the Health Care Financing Administration to maintain a mean LOS of > 25 days. These facilities are most often free-standing hospitals, which may have their own ICUs. Called "regional weaning centers" in Table 9, they serve several to many hospitals in their geographic areas. (2) Step-down units or noninvasive respiratory care units have no specific LOS requirement. These units usually reside within a host hospital and primarily serve that hospital. While both settings have acute-care staffing, but not critical care (ICU) staffing, they are often dissimilar in hospital admission and discharge criteria, treatment capability, and the availability of specialty/subspecialty consultation services and procedures offered on site, all of which may have a significant effect on outcome of care. Both of these types of facilities are characterized by less intensive staffing and less costly monitoring equipment, and, therefore, they generate less cost per patient than do ICUs.[219,220]

Recommendation 12. *Weaning strategy in the PMV patient should be slow-paced and should include gradually lengthening self-breathing trials.*

Rationale and Evidence (Grade C)

Despite differences in patient population and physical facilities, the available information on strategies for discontinuation of PMV in the studies listed in Table 9 has a number of similarities (Table 10). Daily SBTs initially are uncommon since patients have already established themselves as very unlikely to "turn around" in 24 hours. Instead, ventilator support is gradually reduced, using common modes of partial support (Table 7). Usually at the point of approximately half-support, patients are switched to the SBT approach described above, which often is employed along with self-breathing trials of increasing duration. Since most patients are tracheotomized, tracheal collars are used, instead of the familiar T-piece in the ICU, to supply oxygen and humidity. During these procedures, it is important for the staff to remain patient. Psychological support and careful avoidance of unnecessary muscle overload is important for these types of patients.

Long-term facilities may be a particularly useful place to implement protocols such as described earlier in the "Managing the Patient Who Has Failed an SBT" section (Recommendation 8).[221–223] Skilled nonphysician personnel (eg, registered nurses and registered respiratory therapists) are generally present in these units continually and, thus, are in a position to constantly interact with the patient

and to make ventilator adjustments as appropriate. Of interest, after the implementation of a therapist-implemented protocol for weaning, the time to wean in a regional weaning center, with its predominantly "medical" population, declined from a median of 29 days to < 17 days over a 2-year period of protocol use.[223]

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Sudden Development of Right and Left Lung Asymmetry in a Pediatric Patient Following Craniotomy

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A 7-year-old girl presented to the pediatric intensive care unit following a craniotomy that left her with dysphagia, poor cough, and problems with retained secretions. Pulmonary function and blood oxygen saturation worsened for 3 days after surgery. Noninvasive positive-pressure ventilation and increased fraction of inspired oxygen improved oxygenation. Glycopyrrolate was administered to decrease secretions but had little effect. The first chest radiograph showed left lung hyperinflation. The right lung showed loss of volume and elevation of the right hemidiaphragm. There was no mediastinal shift. Another chest radiograph 3 hours later showed substantial improvement. We discuss the causes of acute lung volume asymmetry and possible interpretations of the radiographs.
Key words: lung asymmetry, pediatric, glycopyrrolate. [Respir Care 2002;47(1):91–93]

Introduction

A 7-year-old girl presented to the pediatric intensive care unit postoperatively following a retromastoid craniotomy for resection of a malignant ependymoma of the fourth ventricle. This was the fourth surgery for resection of this tumor, which was diagnosed by MRI. The patient was extubated on arrival to the pediatric intensive care unit. Cranial nerve dysfunction from the surgery resulted in oral-motor difficulties, including dysphagia, inability to mobilize secretions, muscle weakness, and ineffective cough. These complications resulted in re-intubation on postoperative day 2. Following a short-term regimen of intravenous glycopyrrolate (instituted in an attempt to decrease the excessive volume of secretions), the patient was again extubated on day 3. Subsequent oral and nasotracheal suctioning was required every 30–60 minutes for copious, viscous (nonpurulent) secretions.

Over the next 12 hours, the patient required an increase in fraction of inspired oxygen, from 35% to 50%, and it was difficult to maintain oxygen saturation > 85%. Tachypnea, nasal flaring, shallow respirations, and muscle weakness developed, and impending respiratory failure was apparent. Noninvasive ventilation was instituted with bi-level positive airway pressure at an inspiratory pressure of 16 cm H_2O, an expiratory pressure of 8 cm H_2O, and a 5 L/min oxygen bleed-in. Oxygen saturation improved to 92%. That evening the patient was changed to supplemental oxygen by mask. Suctioning requirements again increased to every 30 minutes throughout the night. Seventeen hours following extubation, a chest radiograph (Fig. 1) was obtained to evaluate the presence of infiltrate or atelectasis.

The patient was maintained on supplemental oxygen by mask, and oxygen saturation continued to improve, to 95%, without any further increase in oxygen requirement. A second chest radiograph (Fig. 2) was obtained 3 hours later. Compare Figure 1 and Figure 2.

Questions

1. What is the most obvious change?
2. What can account for the dramatic change in the chest radiograph in Figure 2?

Discussion

Figure 1 reveals asymmetry of the right and left lungs, with hyperinflation of the left lung, loss of right lung volume, and elevation of the right hemidiaphragm, but without mediastinal shift. Normally, lung volumes can be evaluated by observing the presence of lung tissue extend-

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Fig. 1. Chest radiograph 17 hours after extubation.

Fig. 2. Chest radiograph 20 hours after extubation.

ing out to the periphery and diaphragmatic excursion extending to the level of the 9th to 11th ribs posteriorly.[1] The left hemidiaphragm is normally located lower than the right because the presence of the liver in the right upper quadrant of the abdomen elevates the right hemidiaphragm slightly. Acute discrepancy in the lung volumes, as seen in Figure 1, is a cause for concern. Decreased lung volumes may be attributed to the presence of retained secretions resulting from ineffective cough, immobility, or shallow inspiration commonly seen in postoperative patients.[2] Decreased lung volumes accompanied by opacities illustrate the presence of atelectasis. Decreased lung volumes and elevation of the diaphragm on the affected side, with mediastinal shift toward the opacity, may indicate atelectasis caused by bronchial obstruction.[3]

Increased lung volumes caused by sudden expansion of one lung may result from pleural effusion, which may present with air bronchograms if the underlying lung is atelectatic.[3] In Figure 1, ball-valve obstruction or pleural effusion are unlikely, since there is no mediastinal shift in either direction, nor air bronchograms in the left lung.

In Figure 2 the left and right hemidiaphragms are at the same level. The most obvious change is the prompt and complete resolution of the disparity of lung sizes. Evaluation of diaphragm paralysis should have been considered in the chest radiograph interpretation, based on the patient's recent history of craniotomy and the lung asymmetry seen in Figure 1. Phrenic nerve palsy should be suspected if the affected diaphragm is elevated, mediastinal shift occurs during inspiration, and paradoxical diaphragmatic movement of the affected diaphragm is noted on inspiration during a forced expiratory maneuver or sniffing. The "sniff test" is done under fluoroscopy.[4] This patient was not evaluated in this manner because of limitations of head positioning and inability to cooperate and follow commands.

The most likely explanation for the disparity of lung volumes between Figures 1 and 2, in the absence of mediastinal shift, is transient atelectasis of the right lung. The history of dysphagia, inability to handle secretions, ineffective cough, and muscle weakness support this diagnosis. Airway clearance techniques were indicated to mobilize and facilitate the removal of secretions. It seems less likely that there was transient paralysis of the right diaphragm due to phrenic nerve injury or other damage during surgery, because the lung volumes returned to normal within 3 hours.

Initial interventions used in an attempt to assist in the removal of secretions included nasotracheal suctioning and glycopyrrolate. Frequent stimulation of the bronchi, larynx, or pharynx can result in stimulation of the cough reflex.[5] Repeated irritation caused by frequent suctioning puts the patient at risk for complications such as bleeding, bronchospasm, and irritation of the soft tissues, resulting in swelling, nosocomial pneumonia, increased intracranial pressure, and exacerbation of cerebral edema, which were relevant to this case.[5] Glycopyrrolate was used to control the volume of secretions, but with little effect in this case. The Vest Airway Clearance System (Advanced Respiratory, St Paul, Minnesota) was used to enhance airway clearance following the chest radiographs.

The patient required reintubation the following day. Tracheostomy and gastric feeding tube were later performed to facilitate long-term needs for airway clearance and feeding.

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Letters

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Strong Ion Difference: Metabolic Acidosis or Alkalosis?

I hope you will be so kind as to enlighten me about a statement in an editorial published in RESPIRATORY CARE, "The Strong Ion Difference Approach: Can a Strong Case Be Made for Its Use in Acid-Base Analysis?" (Respir Care 1999;44(1):26–28). The following paragraph from that article is difficult to understand:

> Likewise, the discerning reader will note a parallel similarity with SID [strong ion difference] and the classic anion gap, which are just offset by about 25 mEq/L, which is the sum of the HCO_3^- concentration and the difference between the other normally circulating strong ions (Ca^{++}, Mg^{++}, lactate, urate, sulfate) that are not included in the anion gap calculation. Therefore, an SID greater than 40 suggests a possible metabolic acidosis of either endogenous organic acid overproduction or under-clearance (lactic acid or ketoacid), intake of exogenous acid, or acid precursors (eg, methanol and salicylates). A nonrespiratory, normal SID acidosis suggests a hyperchloremic acidosis from loss of alkaline equivalents, and an SID lower than 40 is consistent with a metabolic alkalosis or a surfeit of unmeasured cations (as in cationic paraproteinemias or ingestion of cations, such as Mg^{++} and Ba^{++}).[1]

I think that SID $>$ 40 is associated with metabolic *alkalosis* and, conversely, SID $<$ 40 is associated with metabolic *acidosis*, but the latter passage says just the opposite. SID is the same as buffer base described by Singer and Hastings in 1948.[2] By definition, buffer base is increased in metabolic alkalosis and decreased in metabolic acidosis.

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The author responds:

Dr Roncoroni is entirely correct in catching my mistake. An SID greater than normal is evidence for a metabolic *alkalosis*, and vice versa. I thank him for pointing this out.

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Reviews of Books and Other Media. Note to publishers: Send review copies of books, films, tapes, and software to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104.

Books, Films, Tapes, & Software

Asthma's Impact on Society: The Social and Economic Burden. Kevin B Weiss, A Sonia Buist, Sean D Sullivan, Editors (Lung Biology in Health and Disease, Volume 138, Claude Lenfant, Executive Editor). New York: Marcel Dekker, 2000. Hard cover, illustrated, 390 pages, $125.

In this 138th volume of the Lung Biology in Health and Disease series, 26 internationally recognized experts describe the social and economic impact of asthma in a comprehensive, well-organized monograph. The first 9 chapters address the social impact, encompassing effects on quality of life, work, psychosocial factors, and sociodemographic interactions, including socioeconomic status. Chapters 6 and 7 provide useful descriptions and comparisons (with comment) of various instruments for measuring quality of life for children and adults. The final 6 chapters describe the economic impact of asthma, with analysis of the complexities and limitations of studies of direct and indirect costs and considerations essential to development and critical understanding of pharmacoeconomic studies. Chapter 12 provides useful examples of several pharmacoeconomic studies of pharmacotherapy, and Chapter 13 discusses details of studies of strategies for preventing asthma.

The editors have achieved their purpose of providing a state-of-the art examination of the social and economic impact of asthma in developed countries, but with little if any reference to its impact in less developed countries, where asthma may be less frequent but also less easily managed because of lack of resources.

This volume will appeal not only to clinicians but also to health policy analysts, health economists, public health officials, managed care administrators, medical students, respiratory therapists, nurses, and anyone else interested in management or study of asthma. It will be most useful as a reference.

Nearly all statements are accurate, except for several assertions that death rates from asthma are "increasing steadily" (page 291, for example); they have been relatively stable in the United States since 1988 and have been decreasing in several other countries. Assertions of continued increases in prevalence of asthma in the Untied States are based on data only through 1994 (pages 33 and 56). A suggestion that use of insecticides to control dust mites might be helpful (page 292) disagrees somewhat with the 1997 National Asthma Education and Prevention Program Guidelines, which recommend only physical options for routine control.[1]

There are very few typographical errors. One that may be confusing is the assertion that "Death rates from asthma in the United States are significantly higher among people living in less affluent countries" (page 291). The authors probably meant less affluent counties or neighborhoods. Data from the International Study of Asthma and Allergies in Childhood generally indicate the lowest prevalences of asthma in less affluent countries.[2]

There was only one word not recognized by my dictionary: "operationalized" (page 35). A more frequent problem is the numerous nonstandard abbreviations, usually defined at first use in each chapter, but "PAQLQ" (Pediatric Asthma Caregiver's Quality of Life Questionnaire) (Page 91) and "FSQ-S" (Functional Status Questionnaire) (page 106) were not defined. Readers using the volume as a reference should not be required to scan the rest of the chapter for definitions of abbreviations. Perhaps a table of abbreviations for each chapter or a glossary at the end of the book should have been considered.

MEDLINE and EMBASE are acronyms that should not be expressed with lower case letters, as they are on pages 103 and 130.

The book is unusual in providing an index to authors whose publications are cited in the text. On the other hand, the subject index is somewhat incomplete—an important deficiency in a text most likely to be used for reference. Additional entries or citations might have included: adherence 201, 203, 257; allergens: cat 204, cockroach 204, 226–7; dust mite allergen 204; asthma: classification 36, fatal 196–7, 200–1, mortality 223, nearly fatal 194, 196, prognosis 42, risk factors 224–9, self-management 202–3; copyright of questionnaires 91; emergency care 206; fireplaces 62; gas stoves 62; perception of airway obstruction 194–5; and psychopathology 205.

This monograph is attractively bound, consistent with previous volumes of this series, and printed with clear, easily readable typeface. It should be a welcome addition to the libraries of anyone interested in asthma and its management.

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REFERENCES

1. National Asthma Education and Prevention Program Expert Panel Report 2. Guidelines for the diagnosis and management of asthma. Available at http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm. Accessed Oct 23, 2001.
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Asthma and Rhinitis, 2nd edition, 2 volumes. William W Busse MD and Stephen T Holgate MD, Editors. Foreword by Charles E Reed. Oxford, UK: Blackwell Science, 2000. Hard cover, illustrated, 2,014 pages plus index, $395.

On initial presentation, this comprehensive work (2,014 pages) on asthma and rhinitis can be somewhat intimidating by its sheer volume of information. Though these diseases have been recognized for centuries, their description and definition continue to evolve with advances in various scientific techniques, particularly at the molecular level. This 2-volume book attempts to comprehensively cover all aspects of 2 complex and frequently intertwined disorders (rhinitis and asthma), starting with a broad overview of their epidemiology, classification, genetics, and pathology. Subsequent chapters then start the breakdown, discussing the disease process from a cellular level, including discussions on neuroregulation and mediators of inflammation.

The book is divided into 2 volumes and 23 parts. The first volume deals with introductory issues, including definitions, classification, epidemiology, genetics, physiology, and pathology, and moving on into discussion of asthma and rhinitis at the cellular level, covering cell types, neuroregulation, mediators of inflammation, restructuring, and remodeling. Volume 2 focuses on the role of allergens and other environmental factors, clinical aspects of asthma and rhinitis in adults and children, and the drugs available for treating these disorders. This volume is geared more toward the clinician who treats asthma and rhinitis on a daily basis. However, this is not just a pedestrian recital of definitions and algorithms on how to diagnose and treat these disorders; included are in-depth discussions on the pathophysiology and mechanisms of symptoms, including cough, airway hyperresponsiveness, and airway inflammation, for various types of asthma, including aspirin-sensitive asthma, nocturnal asthma, and intrinsic asthma.

The material is comprehensive but well written. It is remarkable and a tribute to the editors that despite the long list of impressive contributors the book flows well, as if written by one author. Both volumes are easy to read for anyone with a background in pulmonary medicine, allergy, or otolaryngology. The chapters are comprehensive but concise, well organized, and well written. The diagrams and tables are largely in black-and-white, but are clear and easy to follow.

The 2 volumes are in green covers, well bound, and with print and paper of good quality. There are a total of 230 contributors; all leaders in their respective fields. A large index simplifies looking up specific aspects of the disease processes. Unlike most textbooks today, this one does not include a CD-ROM, which I believe should be a consideration in future volumes.

It is suggested that this book is for the busy physician wishing to keep up to date with the latest information regarding these 2 disorders, but the book appears to be geared more toward those involved in basic or clinical research in asthma or rhinitis. Pulmonologists and otolaryngologists who treat patients with the more complex forms of asthma and rhinitis would benefit most from this book. Anyone wishing to understand asthma from the molecular level would also find this a good reference tool.

The book's organization is logical, with each volume and chapter discussing the material in broad terms, then moving on to more focused discussions. In the first volume, Part 1 deals with the epidemiology of asthma and rhinitis, followed by a discussion of the genetics, anatomy, development, physiology, and pathology, before delving into topics such as eosinophils, lymphocytes, and neuroregulation.

The format of the chapters follows that of the parts, with the first 2 or 3 chapters in each part being introductory and the following chapters applying the topic specifically to asthma and rhinitis. For example, the part on eosinophils begins with a chapter on the development, maturation, and function of eosinophils, followed by a chapter on the mechanisms of eosinophil activation, followed by a discussion of eosinophil mediators, and then applying those concepts to asthma and rhinitis. Each chapter is fairly short (3–8 pages), but inclusive and with extensive references.

Volume 2 primarily addresses clinical aspects and is also written in a toned-down form, beginning with Part 15, which discusses the role of allergens in airway disease, once again beginning with aerobiology in general and the molecular biology of allergens, followed by a discussion of the house-dust mite (a very common allergen among asthmatics), followed by discussion of the role of those allergens in asthma and rhinitis. Subsequent parts discuss the smooth muscle response, exercise-induced asthma, infection mechanisms, and the various mechanisms of asthma and rhinitis drugs. I particularly appreciate that not only is there a part on the clinical management of asthma, but there is also a specific section dealing with asthma in special circumstances. That part addresses topics such as asthma in pregnancy, surgery, the intensive care unit, recalcitrant asthma, anaphylaxis, and asthma in the elderly, all of which are important issues, the specific nuances and management of which are discussed. It is not just a cookbook approach; it also includes a brief discussion of the thought process behind the recommendations, giving one a logical approach to treatment. For example the chapter on recalcitrant asthma discusses the initial considerations in patients with recalcitrant asthma, such as correct diagnosis, compliance, adequate therapy, inadequately treated factors such as allergen exposure, gastroesophageal reflux, and chronic irritant exposure, followed by concise but detailed discussions of each of those issues.

The authors also clearly divided the management of childhood asthma and adult-onset asthma, recognizing the different considerations in each group, including the use of steroids in children, compliance, and the natural history of the disease in children versus adults.

This book is clearly a reference tool that has trimmed any unnecessary "fat" and yet comprehensively addresses airway allergic disease. I would suggest that that Volume 2 would be of greatest value to the clinician and Volume 1 of greatest value to the scientist. For a topic in which the information seems to grow logarithmically, the authors attempt to comprehensively cover all current information. Clearly, the time commitment for a project of this nature, with so many authors, does not allow most textbooks to be truly current, but this book is an excellent reference tool for both the clinician and scientist. The editing is excellent and there are few typographical errors or omissions.

One of my primary criticisms involves the sections on rhinitis. It appears that the book is really geared toward asthma and that the sections involving rhinitis are more of an "afterthought," in terms of the percentage of the discussion that is devoted to rhinitis. I suggest that in future editions the editors would do well to add a part on clinical management of rhinitis, including discussions on recalcitrant rhinitis—a topic that would of value to the clinician.

Despite that criticism, the book as a whole is an excellent reference tool for what is sometimes mistakenly seen as a common and easily treated and easily controlled disease. I applaud the editors on excellent editing, given the 230 authors and 133 chapters. I appreciate the logical sequencing and the clear writing, which make it easy to follow for either the clinician or scientist. The subject matter is obviously very focused and therefore has a somewhat limited audience, but the book would make an excellent addition to the library of anyone seriously involved in the treatment, management, or research of asthma, including pulmonologists, immunologists, allergists, or otolaryngologists. The second volume provides excellent reviews and approaches to treatment for nurses, fellows, residents, or respiratory therapists interested in more detailed aspects of the diseases and their management. Some of the science may be too detailed for those

without substantial background, but the clinical chapters as a whole should be quite clear to anyone who is involved in day-to-day treatment and management. I would use the book to understand in more detail the pathogenetic mechanisms of asthma and rhinitis and, in particular, the clinical chapters to review the treatment and management of more difficult patients.

This book could be improved by the addition of a CD-ROM, which would allow quick access to information and the ability to move rapidly from one topic to the next.

Most textbooks are relatively out of date by the time of publication. Given that asthma and rhinitis are so prevalent in our population, and despite our recognition of these disorders for over 40 years, we still have a lot to learn, as shown by the astronomical increase in information in just the last 5 years alone. Despite this, I would highly recommend this book to anyone who desires to increase or improve his or her knowledge of these diseases on either a scientific or clinical basis. The clinical section could have been more detailed but is more than adequately covered. I find most reference books rather dry and difficult to follow in a logical fashion if one is interested in learning about a topic from *A* to *Z*. Not so with this text. I must admit I was pleasantly surprised by the flow and sequencing of the chapters and parts.

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New and Exploratory Therapeutic Agents for Asthma. Michael Yeadon and Zuzana Diamant, Editors. (Lung Biology in Health and Disease, Volume 139. Claude Lenfant, Executive Editor). New York: Marcel Dekker. 2000. Hard cover, illustrated. 494 pages. $185.

Asthma has always been a fascinating disorder, and there is an increasing incidence of asthma all over the world each year. Though people have spent a lot of time and resources on asthma research to better understand the pathophysiology and to find better control of it, there is still a lot to learn. New data is coming up in many published sources, Web sites, and conferences each year. This 139th volume of the Lung Biology in Health and Disease series involved as authors a very large group of well known, expert researchers, scientists, and physicians, and the book covers detailed, state-of-the-art information from extensive literature reviews. The book will attract health care professionals who have special interest in this field.

The book is subdivided into 3 parts, the first of which includes 4 chapters devoted to the basic science of asthma epidemiology and pathophysiology, which is very thoroughly researched and summarized and will be very useful for physicians or researchers in pulmonary and allergy fields.

The second part includes the next 8 chapters, which cover asthma therapies, including the currently common ones (inhaled β_2 agonists and inhaled corticosteroids), newer agents (leukotrienes antagonists), and those still undergoing investigation (platelet-activating factor antagonists, tachykinin receptor antagonists, phosphodiesterase inhibitors, and immunotherapy with antibody immuglobin-E). This part provides not only the basic science knowledge and applied research that led to the development of each therapy but also addresses the clinical trials of each therapy. This part will be valuable for all health care professionals working with asthma patients.

The chapter on tachykinin receptor antagonists is well written and covers the pharmacologic properties of tachykinin receptor antagonists, reviewing their contribution to tachykinin receptor classification. Data from animal and clinical studies are discussed. The chapter on leukotriene modulators is also well written but comments too little on the possible link with Churg-Strauss syndrome. We also enjoyed reading the chapter on phosphodiesterase type 4 inhibitors and activity of T cell subsets in allergic asthma.

The last 2 chapters in the third part address the future of asthma therapy and trends in clinical trials in asthma. This part gives the current prevailing data on asthma therapy and may even be a good source of new research ideas for asthma researchers.

This book is not a text for readers seeking the results of asthma studies in general, but rather focuses on the latest treatment strategies for asthma, including issues in the genetics and molecular biology of asthma. The book also focuses on current approaches to establishing novel, commercially viable asthma therapies. This book is well referenced, with a generally up-to-date bibliography.

One minor criticism is that some chapters have few illustrations, such as the sections on pathology and pathophysiology of asthma, and some have only occasional tables.

Overall, the book is well written and thoroughly referenced. It provides up-to-date, easy-to-read, and high-quality coverage of new developments in asthma pharmacotherapies, which will be useful to pulmonologists, immunologists, allergists, junior doctors, medical students, and allied health professionals. We highly recommend this book for libraries and enthusiastic asthma care professionals.

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Asthma Sourcebook. Annemarie S Muth, Editor. (Health Reference Series). Detroit: Omnigraphics. 2000. Hard cover, illustrated. 611 pages. $78.

There never seems to be enough information about asthma. Every year there are new and exiting changes in the treatment, monitoring, and understanding of this disease, and many of the changes, especially in pharmacology, can be old news by the time they make it to print. The **Asthma Sourcebook**'s goal is to provide a compilation of current information about the diagnosis of the disease, reports on statistical data, reports on research and prevention measures, and new treatment modalities.

The book is divided into 8 segments that encompass 66 chapters, a glossary, and a resources section. The sections are: "The Nature of Asthma and Related Ailments," "Asthma Statistics," "Theories About the Causes of Asthma," "Triggers and Risk Factors Associated with Asthma," "Diagnosis and Treatments," "Asthma Management," "Other Aids to Wellness and Prevention," and "Additional Help and Information." The book's cover states that it contains "basic consumer health information about asthma," so its intended audience is the consumer and not the medical professional. And there is no disclaimer in the introduction that challenges that conclusion. I read the book as a consumer, but with the eye of a respiratory therapist. The **Asthma Sourcebook** is one

of a series of books in the Health Reference Series. Annemarie Muth, on whom the book gives no background information, edited the book. In addition there are 5 medical editors, all of whom are librarians from public, academic, and medical libraries.

In Chapter 1, "What Is Asthma?" there is discussion of over-the-counter short-acting bronchodilators, which are described as useful in the temporary treatment of mild asthma attacks. The chapter goes on to note that over-the-counter bronchodilators are readily available, which makes them a logical "stop-gap" for patients who do not have medication on hand when an attack occurs. That statement is illogical and stands in the face of best practice. It is precisely the use of over-the-counter medications that leads patients to choose not be under the care of a physician. Rather than over-the-counter medications, short-acting bronchodilators such as albuterol should be the drug of choice for acute asthma. The book goes on to say that patients who use over-the-counter drugs should still see a physician about the long-term control of asthma. Those are irresponsible recommendations and do not agree with the asthma guidelines presented by the National Asthma Education and Prevention Program Expert Panel.[1]

Chapter 5, "Rhinitis Update: A Guide to Diagnosis and Treatment," provides the reader with some solid recommendations about trigger remediation techniques. Triggers are a major component that must be addressed if asthma is to be adequately controlled. Another (albeit short) chapter is dedicated to cardiac asthma and emphysema. This is a common misdiagnosis. Heart failure is often the source of wheezing that some physicians may be inclined to treat with bronchodilating drugs. It is the keenly aware respiratory therapist who can recognize that these asthma-like symptoms are actually an exacerbation of heart failure.

Chapter 12, "Asthma by Race, Sex, Age, and State," was 34 pages long and was too lengthy. Over half of the chapter is taken up by graphs that detail a historical and current perspective on asthma death rates, average number of office visits, emergency department visits, and hospitalizations. To the typical consumer this is information overkill. Chapter 14, "Seasonal Variation of Hospitalizations for Asthma, Bronchitis, and Emphysema," is a thorough description of a Kaiser Permanente study of a health-maintenance organization in Portland, Oregon. Though that information may be helpful to those who reside in the Northwest, its details mean little to the asthmatic in Biloxi, Mississippi, or most other parts of the country. Chapter 17, "Mortality and Markers of Risk of Asthma Death," were useful to me but would be of little interest to the layman. The chapter was written as a scientific paper, and the statistical formulas would be of no value to the untrained or uninterested.

Most of the remaining chapters are compilations or excerpts from medical studies or chapters from other books, and this book attempts to offer a "Reader's Digest" version of those articles. Sometimes they hit the mark; other times they are way off. An example of the latter is Chapter 24, "Interleukin 11 and Asthma." This 4-page chapter attempts to describe the cellular events that occur in the mediation of lung injury. Terms such as *enzyme-linked immunosorbent assay* and *mycobacterium avium-intracellulare* are not likely to be helpful to a parent wishing to understand airway hyperresponsiveness.

Chapter 26, "Targeting Asthma Triggers," provides good common sense and achievable steps to reduce or eliminate asthma triggers. I also found Chapter 29, "How Energy Policies Affect Public Health," most interesting. Providing the consumer a historical perspective on why we have many of our current public health problems will better enable us to fight for improved quality air. Another useful chapter is Chapter 38, "Access to Asthma Health Care by African-American Families," which offers some excellent case studies describing the barriers that that population of asthmatic patients deal with. A detailed review of family perspectives on access to asthma health care is presented. These case studies describe the cultural and socioeconomic factors that can act as a huge barrier to best care for a population that so desperately needs it.

Chapter 40, "Asthma Medications," left out many of the newer drugs. This chapter is based on the asthma guidelines,[1] which were published in early 1997. Many newer medications have been introduced since then. There are 2 chapters in the **Asthma Sourcebook** that describe the important role of the allergist and pharmacist in asthma care, but, unfortunately, there is no mention of the front-line caregivers: nurses and respiratory therapists. If role delineation was the intention, those 2 essential medical professionals should have been mentioned.

The glossary and reference sections were both well researched and offer the reader much-needed additional information. As I finished the book I was struck by the realization that I had many more questions than answers. If the intended reader was the layman, the publisher was off the mark. Though there are some chapters written at the level of the layman, most were obviously directed toward the medical professional. When developing educational materials for the public, it is essential to realize that most patient-directed literature should be written at the fourth-grade level. That was accomplished in only a few chapters. The cost of this 642-page book is $78. When you consider that a majority of asthma patients are of lower socioeconomic profile, the cost of the book may be prohibitive to many patients and families, who, fortunately, can get much of the same information for less or even for free. Neither would I recommend this book for the medical professional, as it contains some erroneous and outdated information that is counter to the current best practices of asthma care.

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REFERENCE

1. National Asthma Education and Prevention Program Expert Panel Report 2. Guidelines for the diagnosis and management of asthma. Available at http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm. Accessed Oct 23, 2001.

Combination Therapy for Asthma and Chronic Obstructive Pulmonary Disease. Richard J Martin and Monica Kraft, Editors. (Lung Biology in Health and Disease, Volume 145, Claude Lenfant, Executive Editor). New York: Marcel Dekker, 2000. Hard cover, illustrated, 346 pages, $150.

The Lung Biology in Health and Disease series, edited by Claude Lenfant, has been a remarkable chronicle of pulmonary medicine. With this most recent addition, which reviews combination pharmacotherapy in asthma and chronic obstructive pulmonary disease (COPD), at this writing this series comprises 145 volumes, encompassing the evolution of thinking over the past 2–3 decades, on the entire spectrum of respiratory diseases and disorders. The appreciative

reader is delighted to learn that further exciting additions to this series are in the planning stages. This particular volume, edited by Martin and Kraft, complements earlier monographs on COPD, asthma, and airway pharmacology. The editorial approach taken to reviewing combination pharmacotherapy can conveniently be divided into 3 sections.

Section I includes Chapters 1 through 5. Each chapter reviews an individual class of pharmacologic agent used in treating asthma and COPD. The pharmacologic classes included in this section are corticosteroids, β_2 adrenergic agonists, theophylline, leukotriene modifiers, and anticholinergic bronchodilators. Subheadings in these 5 chapters have similar approaches, emphasizing historical perspective, pharmacologic efficacy, and clinical efficacy. Another class of drugs that might have been included in this section is mucoactive products. Both asthma and COPD involve increased mucus production as an important pathophysiologic component of airway obstruction. Extensive work has focused on developing therapies for excess mucus production. Most of this work has been unsuccessful, but intriguing information from studies with such diverse products as surfactants and macrolides suggests that mucoactive products might play a role in managing asthma and COPD. Combination therapy with mucoactive drugs and bronchodilators might prove to be of clinical value. Also of potential value would have been analyses of actual patterns of single and combination drug use by physicians in treating asthma and COPD. Especially valuable would have been comparisons of actual drug use for these diseases among physicians in the United States, Europe, and less developed countries. The absence of that information in this book is especially striking given the comments about theophylline in the only chapter of this book written solely by Europeans: Dent and Rabe state on several occasions that theophylline "is now one of the world's most prescribed therapies for asthma and COPD." For American physicians that is probably a surprising statement, as recent trends have been to limit the use of theophylline. Perhaps use of theophylline is more prevalent in the United States than I suspect, or perhaps the use of theophylline might be variable around the globe. In today's world of physician profiling by pharmaceutical companies, such data on drug prescription, both individual and combination, in asthma and COPD should be available.

Section II comprises a single chapter on the influence of circadian rhythm on the efficacy of asthma medications. Chronotherapy is becoming an especially important issue in asthma management, for 3 reasons. There is now the potential for administering inhaled corticosteroids once daily, which might minimize systemic adverse effects of inhaled corticosteroids but might also result in reduced efficacy. The availability of long-acting theophylline preparations provides additional options for simplified symptom control. The growing recognition of nocturnal symptoms as an important clinical feature of asthma emphasizes the value of using long-acting bronchodilators at night. Surprisingly, there was no reference in this chapter to once-daily use of leukotriene modifiers. An intriguing and little-understood issue is the explanation for favoring their use in the evening rather than in the morning.

Section III includes the final 2 chapters, which address, in turn, the combination therapy for asthma and COPD. Kraft summarizes the extensive asthma research literature and the many different approaches to combination therapy. Limiting this analysis was the difficulty in many of the studies performed to define baseline asthma severity and to estimate the effect of adding additional agents as asthma severity worsened. This important consideration makes it difficult to apply Kraft's summary to general recommendations for combination pharmacotherapy based on the generally accepted stepped-care approach to asthma management. Sutherland and Martin review selected aspects of combination pharmacotherapy in COPD and briefly discuss such nonpharmacologic treatment approaches as pulmonary rehabilitation and smoking cessation.

This should not be considered a basic textbook for students of pulmonary disease. The price probably puts this book outside the budget of many asthma and COPD clinicians and students. The target audience is probably a select group of respiratory physicians interested in both treating patients with obstructive airways disease and investigating asthma and COPD therapies. For that audience, this monograph may serve as a useful additional information source on the wide array of clinical studies performed in this field.

The book is well produced. The typeface is easily read and quite clear. There are very few tables, but the figures are nicely reproduced from original publications. Both a subject and author index are included, and these seem to be quite accurate. Extensive reference lists are included at the end of most chapters, but in many chapters the references tend to be from the 1980s and early 1990s. The monograph has a sturdy hard cover, which I found useful. It had the advantage of standing up well, despite being carried aboard many airplanes.

Overall, this monograph provides a useful summary of information for respiratory physicians with special interest in treating or studying asthma and COPD. Although not to be recommended as a primary reference, the information contained in this monograph may complement standard textbooks in the field of respiratory medicine.

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Particle-Lung Interactions. Peter Gehr and Joachim Heyder, Editors. (Lung Biology in Health and Disease, Volume 143, Claude Lenfant, Executive Editor). New York: Marcel Dekker. 2000. Hard cover, illustrated, 802 pages, $225.

Particle-Lung Interactions is the 143rd volume of the long-running Lung Biology in Health and Disease series and represents the latest of only a few volumes dedicated primarily to particle-lung interactions. The text's editors, Peter Gehr and Joachim Heyder, recruited a collection of expert authors from academia, industry, and government to provide a comprehensive, multidisciplinary approach to particle-lung interaction. The focus is on the dynamic levels and mixtures of particles that the respiratory tract sees with every breath and how this may impact health risk. The basis for assessing effect covers a fascinating scientific spectrum, ranging from molecular and cell biology to epidemiologic studies characterizing exposure and effect.

The book is smartly structured for the range of topics, into 6 parts. The first part is an overview chapter. The second part describes the types of particles that interact with the lung. The third section addresses inhalation of particles, including deposition and clearance. Parts 4 and 5 cover a variety of molecular, cellular, and systemic response topics. The final section consists of 3 chapters that address health consequences. As this is not a book aimed di-

rectly at respiratory therapists, the review of these sections will highlight the areas, in my opinion, of most relevance and importance to the Respiratory Care readership.

The single-chapter Part I provides an introductory overview of the entire text by highlighting the critical necessity of bridging two distinct subjects: namely, particle science and lung biology. As stated in the text, the framework is to link toxic particles with the target organ of the lung. This entails much expertise, ranging from sampling strategies of the particulate source to the interpretation of toxicity data and its link to real-world exposure. The discussion of the historical aspects of the scientific studies in this subject area is fascinating reading. The regulatory aspects of particulates are summarized concisely, and the overviews of key topics related to particle-lung interaction (detailed by others later in the book) is helpful. Figures consist of basic black-and-white graphs and drawings, many quite familiar to those working in this subject area. For the practitioner or anyone else interested in an overview, this chapter meets its goal.

The second part of the book consists of 3 chapters describing particles that interact with the lung: specifically, environmental, occupational, and medicinal particles. Though the final chapter on medicinal particles is by far the most directly applicable reading for the respiratory therapist, there were several passages on environmental and occupational particles that were both of general interest and associated with allied health practice. For example, the chapter introduction on environmental aerosols reminds us that, by definition, the entire atmosphere is an aerosol, meaning that it is a 2-phase system consisting of gases and particles (solid or liquid). The discussions on atmospheric particle sizes, sources, and dynamics provide interesting background for a detailed understanding of the role of such particles in climatic pollution and health effects research. Likewise, the chapter on occupational aerosols is informative and interesting, particularly in the descriptions of particle generation in the health care environment, such as the particles produced in a variety of dental and medical procedures, including debris of blood, cells, and tissues, with particle sizes ranging from 0.7 μm to 20 μm. Furthermore, it is described how intact human papilloma virus deoxyribonucleic acid was detected in smoke produced from a carbon-dioxide-laser procedure. Despite few tables and figures, the chapter was very informative.

The chapter on medicinal particles contains an eclectic collection of topics, including aerosols, issues associated with particle distribution, and some statements on receptor distribution. Here the text's style falls short in places with regard to helping the uninitiated reader. For example, it is stated that the nasal route might be used for systemic treatment, but with no mention of why or the role of nasal blood flow. Also, there is some description on the distribution of airway receptors in the lung, but with no mention of the relevance to therapy. Finally, the authors state that descriptions of clinical response to inhaled drugs are of little value in the absence of accurate measurements of the dose at the site of action, only to conclude the section by stating that whether the efficacy is dependent on local dose remains to be clarified. What would be more useful would be a description of how we currently are able to measure "exposure" to medicinal particles (eg, delivered concentration estimating dose, pharmacokinetic) and how this relates to clinical response measured by currently-available techniques. Despite those limitations, other topics are addressed that are often overlooked in such chapters. Specifically, the data on the effects of external resistance on deposition are reviewed, as is the importance of bronchial circulation in aerosol therapy. There are general descriptions of delivery devices and a useful review of various diseases as they relate to aerosol therapy. A brief concluding section on bioequivalence is provided, but there is no direct discussion of the impact of propellants and excipients on particle-lung interaction. A discussion of such issues would have been welcomed.

Part 3, "Inhalation of Particles," is concerned with aspects of deposition, retention, and clearance, and provides discussions on related mechanisms, methods of study, and models applied in the study of aerosol dynamics in the lung. These 3 chapters contain several mathematical formulas that are probably best reserved for discussions in aerosol physics labs; nevertheless, the associated text provides sufficient background to the pertinent issues for practitioners and provides a good working basis for the molecular and cellular biology sections that follow.

Part 4 is concerned with the molecular and cellular responses of the lung to inhaled particles and discusses particles as the stimulus for the lung's biologic response. The 5 chapters in Part 4 discuss the role of cytokines and other mediators and how particulate toxins affect expression of several genes that are the basis of the lung's reaction. As the most characterized of particles, silica is used in most test systems as the stimulus. Chapters specifically apply new information in lung biology to particulate exposure in the context of inflammatory cells' responses, as well as immune response in general and antigen presentation in particular. Thus, those interested in a summary of molecular and cell biology applied to inhalation toxicology of particles will find this well-referenced chapter useful.

Part 5 examines systemic effects from particles, with individual chapters addressing mucus and mucociliary clearance, the role of surfactant, mechanisms of cardiopulmonary effects, and neural reflex activation. Part 6 concludes with a series of chapters on health consequences, all pointing to the associations of respirable particulate air pollution and morbidity and mortality. These chapters discuss work reported up to the last 5 years or so and point out that health effects from particulate pollution (especially smaller particles) are real, with mechanistic underpinnings that are clearly plausible and, hopefully, preventable.

In summary, this textbook is an up-to-date review of particle-lung interaction that reviews existing knowledge in particle science and its application in advances in lung biology and particulate exposure. For the disciplines directly involved, the book is an important compendium. For students both of public health and natural and physical science, it presents a strong blending of disciplines and worthwhile reading. For those in respiratory care, the book was not intended to address therapeutics, and does not; however, it is worth a trip to the library if one seeks further explanation of any of the subjects mentioned above.

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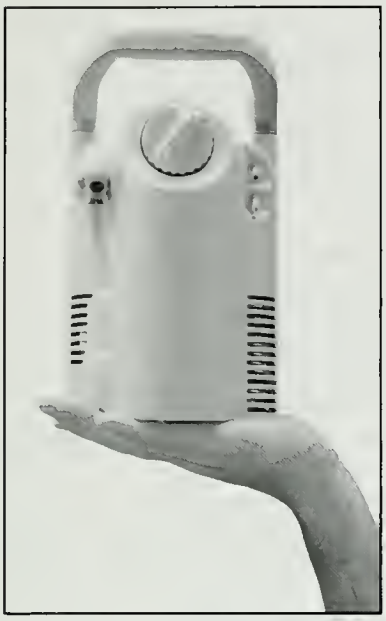

Personal Oxygen System. The *HELiOS* Personal Oxygen System encourages an active lifestyle for long-term oxygen therapy patients, according to HME Technologies. The portable unit weighs only 3.6 pounds filled, enabling patients to wear it, rather than carry it and no canisters or batteries are required. The manufacturer says that the *HELiOS* home reservoir typically lasts four to six weeks between refills. For more information from HME Technologies, circle number 192 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Aerosol Inhalation Monitor. Vitalograph Inc has developed the Aerosol Inhalation Monitor (AIM) to assist RCPs in educating patients in the correct technique to use MDIs to permit otimal medication therapy. The product, according to the manufacturer, displays the patient inhalation flow rate on a needle gauge with a red & green seale. The objective, says Vitalograph, is to keep the needle in the target green section during inhalation. In addition, a green light appears after each suecessfully completed task (firing, delivery, breath-hold), and a red light indicates any difficulties. An optional pediatric incentive device is available. For more information from HME Technologies, circle number 193 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Pediatric Series Suction Regulators. Boehringer Laboratories Inc introduces The New Pediatric Suction Regulator Series. According to Boehringer, these new suction regulators are designed to meet the unique clinical needs of pediatric patients, neonatal ICU and labor & delivery applications by providing safe, low vacuum (0-100 mm Hg) suction to the patient. Model 3710 is a continuous pediatric regulator with on/off selector valve, while Model 3711 is a continuous regulator without the on/off selector valve. Model 3714 is an intermitting pediatric regulator with off/continuous/intermitting selector valve. For more information from HME Technologies, circle number 194 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Allergic Rhinitis Medication Label Change. AstraZeneca LP recently announced that the U.S. Food and Drug Administration has approved revised labeling describing an onset of action within 10 hours for its allergic rhinitis medication, RHINOCORT AQUA (budesonide) nasal spray. The manufacturer says that this labeling ehange was based on results from elinical studies in both seasonal and perennial allergic rhinitis patients. For more information from HME Technologies, eirele number 195 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Aerosolized Medication Device for Infants. WestMed introduces the PediNeb, a new modality in aerosolized medieation delivery. According to WestMed, the PediNeb was designed to enhanee the delivery of aerosolized medication while reducing the apprehension of young patients. The pacifier calms the infant/toddler, while the medication port directs aerosolized medication to the nose. For more information from HME Technologies, circle number 196 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

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Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Scheduled Professor's Rounds 2002

Withholding and Withdrawing Life Support in the ICU — Gordon D Rubenfeld MD / Richard D Branson BA RRT FAARC — Live February 12; Audio March 5

Weaning from Mechanical Ventilation: New Insights, New Guidelines — Neil R MacIntyre MD FAARC/ Dean R Hess PhD RRT FAARC — Live April 16; Audio May 14

Neonatal and Pediatric Ventilators: What's the Difference? — Mark J Heulitt MD FAARC/ Richard D Branson BA RRT FAARC — Live May 21; Audio June 25

Ventilator Graphs: What's with That Wave? — Jon O Nilsestuen PhD RRT FAARC/ Richard D Branson BA RRT FAARC —Live July 16; Audio August 13

Talking with Patients and Families About Death and Dying— Helen M Sorensen MA RRT FAARC/ David J Pierson MD FAARC —Live August 20; Audio September 17

Pressure vs: Volume Ventilation: Does It Matter? — Robert S Campbell RRT FAARC/ Richard D Branson BA RRT FAARC — Live September 10/ Audio October 8

Inpatient Management of COPD — Randall Rosenblatt MD/ David J Pierson MD FAARC — Live October 22; Audio November 12

High-Frequency Oscillatory Ventilation — Thomas E Stewart MD/ Richard D Branson BA RRT FAARC — Live November 19; Audio December 10

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
- — Current job listings
- — American Respiratory Care Foundation fellowships, grants, & awards
- — Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

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Feb. 6–8	West Virginia Society for Respiratory Care's Winter Meeting; **Davis, WV**	Jay Wildt at (304) 357-4851 or jay.wildt@ucwv.edu; or Anna Parkman at (304) 357-4837 or AParkman@ucwv.edu
Feb. 6–8	Oregon Society's Pacific Northwest RC Conference; **Portland, OR**	Tim Mixdorf at (503) 537-1570 or tmixdorf@providence.org
March 13–15	Greater Bay Area Chapter of the CSRC's 23rd Annual Tahoe Conference; **Lake Tahoe, CA**	California Society for Respiratory Care, (925) 866-6643, (925) 947-4487, or www.csrc.org
April 4–5	Alabama Society for Respiratory Care Clinical Conference at the Beach; **Gulf Shores, AL**	Bill Pruitt (251) 434-3405 or wpruitt@jaguar1.usouthal.edu
April 28–29	29th Annual Pacific NW Regional Respiratory Care Conference; **Bellvue, WA**	Ellen Perry (425) 576-1171 or ezperry@earthlink.net
Date	**Other Meetings**	**Contact**
March 4–7	ATS, AARC, Healthsouth Rehabilitation Hospital, and the ALA of Nevada's 21st Annual High Sierra Critical Care Conference; **Reno, NV**	Louise Martin, (775) 829-5864 or lmartin@lungs.org
March 12–13	Practical Spirometry Course; **Rochester, MN**	Mayo Pulmonary Services, Mayo Clinic, (800) 533-1653

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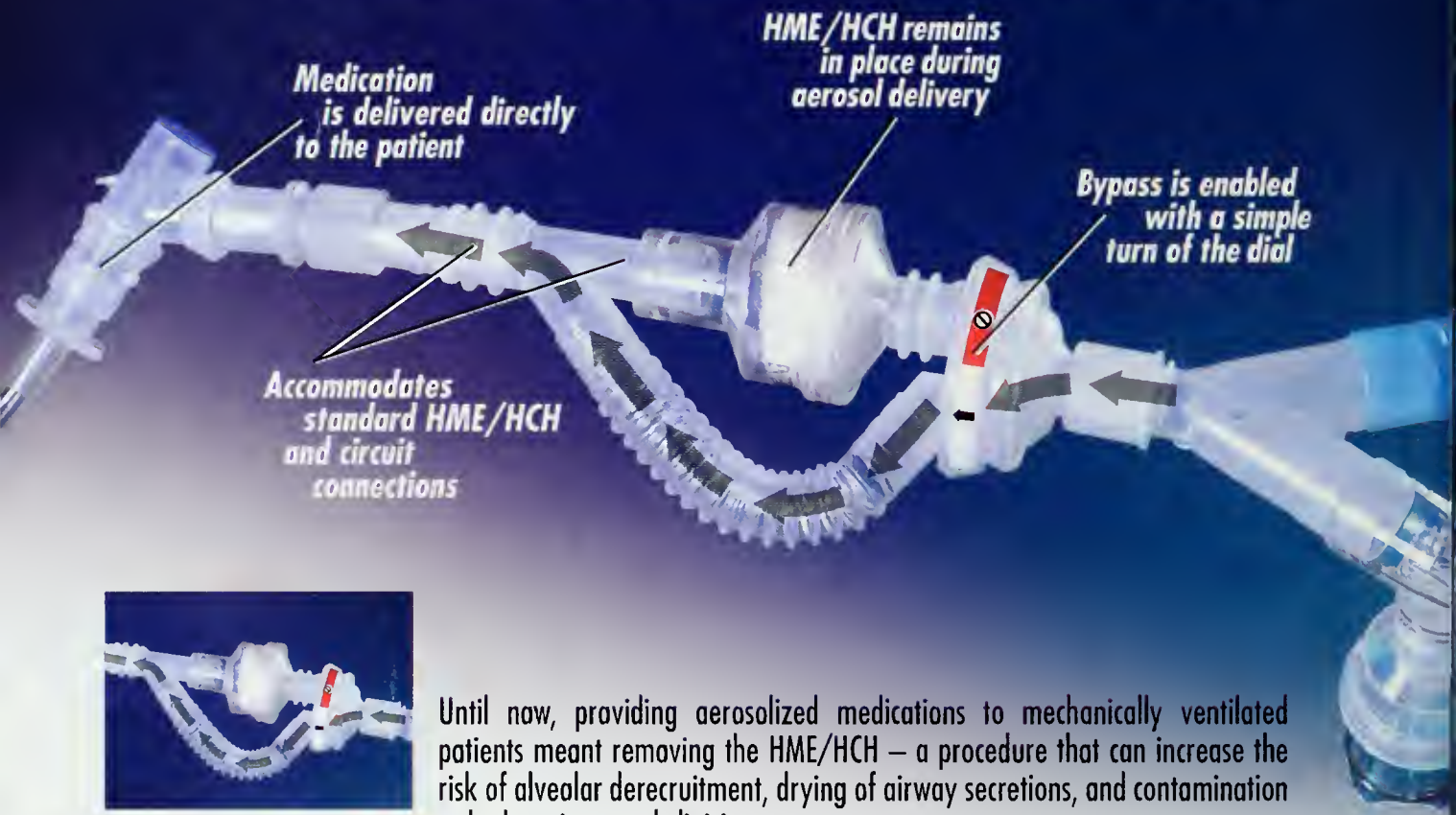
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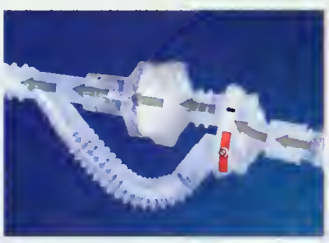

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